

# THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—23RD YEAR.

SYDNEY, SATURDAY, JANUARY 25, 1936.

No. 4.

## Death of His Majesty King George V.

*As this issue of the journal is in the press news comes of the death of His Majesty King George V. The reports that came from his bedside during the last few days of his illness held little hope that he would be spared to the peoples of the Empire whom he served with untiring devotion, and who held him in high honour and affection. The members of the medical profession in Australia join with the whole people of the Commonwealth in appreciation of the life and character of His late Majesty, in sympathy with Her Majesty the Queen and the Royal family, and in unswerving loyalty to the throne.*

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### The Halford Oration.<sup>1</sup>

REMARKS UPON THE CHOICE OF MEDICINE AS A PROFESSION, THE PROGRESS OF MEDICINE AND SOME OF THE METHODS BY WHICH THE PRESENT POSITION HAS BEEN ATTAINED.

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DEEPLY conscious as I am of the compliment paid me when I was invited to deliver this oration, it was with mixed feelings and very considerable misgivings that I accepted the invitation which I received by cable, as our ship was leaving Cape

Town, just a month ago. An occasion such as this calls for one's best, and there were good reasons for my diffidence. A theme must be selected at short notice and there was but little time in which to collect one's thoughts. The choice of subject presented a further difficulty, for, while you gave me a wide scope, I had no knowledge until we landed at Fremantle of the history of the oration, of the titles selected by my predecessors or of the constitution of my audience. I was indeed at sea, and only too well aware that, should the weather prove inclement, and it has been none too kind, one's mental processes might thereby be inconveniently disturbed. Apologia are at times permissible. Divorced from all sources of reference and information a technical address was clearly impossible, and I would crave your indulgence should my remarks savour of platitude and my random discourse prove unworthy of your trust.

<sup>1</sup> Delivered at the Australian Institute of Anatomy, Canberra, on September 16, 1935.

George Britton Halford, whose memory we honour today, must have been a man of outstanding personality, who possessed to an exceptional degree those qualities of mind demanded by the position in which he found himself, when in 1861 he was appointed the first occupant of the Chair of Anatomy, Physiology and Pathology in the University of Melbourne. The organization of the Medical School was a great achievement, but Halford was no mere organizer and administrator, for the titles of his contributions to the literature clearly indicate his interest in, and enthusiasm for, original inquiry. He was not content with the mere study of structure, but, like his great prototype, Sir Charles Bell, whose initial training was also anatomical, he wished to know the reason why. Comparative anatomy appealed to Halford, as it did to Bell, and it is not surprising that the comparison of the hand of man with that of the ape should have engaged the attention of these two students of structure, to whom problems of function were of absorbing interest.

At the expense of a digression I am tempted to refer to two observations which occur to me in passing.

The frequency with which abnormalities of the distal segments of the limbs are transmitted through many generations is a striking fact. Some time ago I met with a mother and her child, in both of whom a digit, with the characteristic three phalanges and musculature of a finger, replaced the thumb. The deformity had long been, so the mother informed me, a feature of the family, and upon referring to Annandale's monograph upon "Deformities of the Hand" (this, to the best of my recollection, is the title of his work), which was published, I think, in 1854, I found that he had traced the abnormality in this particular family through several preceding generations prior to that time. This is an example of a gross deformity, but minor degrees are transmitted with similar frequency. There is a legend of the distant past which tells us that an old woman, regarded no doubt in those days as a witch, when begging alms, was turned from the door of the castle of a Scottish chieftain and that as she went her way she cursed the family and predicted that, to commemorate the scurvy treatment she had received, they and their brood would for ever afterwards have crooked little fingers. And, as a scion of the house told me not long ago, her "prophecy" was fulfilled. "A wise old woman", you say, "and an observer."

The transmission of functional anomalies is perhaps of even greater interest.

I recall the case of a woman with a paralysis of the left arm and leg and loss of speech, seen many years ago. The patient was left-handed, as was to be expected, for it is a well established fact that the speech centres in left-handed people are situated on the right side of the brain. It was not unnatural, however, that her brother should ask me why I had suspected that his sister was left-handed, nor was I surprised when he told me that left-handedness was "in the family". But it was interesting to hear from him that his name was Kerr, that "ker" (I think I am right in saying that the word is spelt with one "r") is the Scotch for clumsy, that the left-handed in former times were often called "ker-handed", and that this he believed was the origin of his surname.

I have since come across an old ballad which tells of a border raid in the olden days, when the Kerrs, a clan of border reavers, smote the English with their left hands. You will find that there is to this day a strong tendency to left-handedness in some branches of the Kerr family. Is it not an arresting thought that a physiological characteristic such as this should be transmitted throughout the ages?

To travel is an education. Three-quarters of a century have passed since Halford first set foot upon these shores. He was no doubt vividly impressed, as one is today when one crosses the wide seas for the first time, with the vastness of the ocean and with the bonds which bind this great Empire of ours together. As a people we have common hopes, common traditions and common sentiments. And Halford, when he first came from "Home", was, I venture to believe, given a welcome in keeping with the extraordinary kindness and hospitality with which you receive your guests today.

We live in a wonder age. But are the discoveries and applications of science to be regarded without qualification as a boon? Where are they leading us? Where is it all to end? Circumstances change with the passing of the years, but adaptability is, I believe, a racial characteristic to which our national success is largely due. We are faced with the world-wide problem of unemployment, for which the elaboration of machinery and over-production are admittedly in large part responsible. Are we on the verge of an era when ability, initiative and personal endeavour are to be discouraged and discounted? God forbid! Let us hope that this will never come to pass.

I would speak of the choice of medicine as a profession and of some of the methods by which the progress of medicine has been achieved—two very different themes, but both after Halford's own heart. Would that I could treat my topic with the distinction it deserves.

The career of medicine, as indicated by the applications for admission to our schools, is nowadays more popular than ever it was, and limitation in numbers is being forced upon our universities if we are to provide adequate facilities for our students. All will agree that every boy of promise should be given the opportunity in life which his capacity merits, but supply and demand must ever be factors which call for serious consideration in relation to the future of the human race, and universal education becomes fantastic when carried to extremes on academic lines which are not adapted to the possibilities of practical application. There was much to be said for the custom in vogue in Scotland in former days, when the village dominie derived his kudos in large part according to the special attention he paid to, and the reputation made by, the lad of promise whom he prepared for the university bursary examination. Nowadays the master's efficiency is rather gauged by the number of pupils who pass through his hands and are successful in obtaining the school leaving certificates. The beneficent intention of the philanthropist by whose generosity the fees of students of Scottish

birth entering the Scottish universities were paid, quite irrespective of intellectual merit and attainment, was, I venture to think, a misguided effort. Many of these lads, who were thus induced to matriculate, with a view to entering the professions, would have been better employed, both in their own interests and in the public weal, had they devoted their attention to vocations to which their qualifications were more suited.

Nowadays, when competition is so keen, the future of our children is a serious consideration. When a boy has definite wishes and holds strong views, and particularly when he shows undoubted capacity for the career he wishes to adopt, he should, if possible, be given all encouragement. But this is the exception rather than the rule; it often happens, indeed, that the boy has no personal inclinations and no obvious qualifications for a particular calling. Parents have sometimes a quite erroneous idea of a boy's abilities and potential worth. The schoolmaster's advice may here be helpful; on the other hand, his opinion may be of little value if it is based, as often happens, upon the boy's achievement in the stereotyped entrance examinations demanded by the universities.

Some of you may have read the biography of Sandison, of Oundle. After Sandison was appointed to the headmastership of this old grammar school, which became one of the great public schools of England, he was concerned to see that some boys remained term after term in the same class, and it occurred to him that it might be the system and not the boy that was at fault. Accordingly Sandison established practical departments, agriculture and engineering, so far as I can recollect, among the number. The result was interesting, for he found that some of the apparent dullards, who had shown no aptitude for or interest in the classics or mathematics, excelled when they applied themselves in these various directions. A venture of this kind has, however, its drawbacks and limitations, for the university entrance examinations constitute the "open sesame" to most vocations.

When, a number of years ago, an elaborate inquiry into medical education was conducted by the Edinburgh Pathological Club, I hazarded the suggestion that there was room for a new profession. A boy about to leave school, who has no settled plans as to his future, might, I think, with advantage, be entrusted for a period to the care of a tutor, a specialist, whose particular duty it would be to size up the lad's ability, capacity and personality with a view to advising his parents as to the walk in life for which he was best fitted.

A variety of reasons may determine the choice of medicine as a profession. An inquiry of the kind would be instructive. Some of our colleagues would tell one that they had been brought up in a medical atmosphere; it might be that their fathers were engaged in a practice to which they had expected in due course to succeed. But many, perhaps the majority indeed, would say that they had had no special leanings towards doctoring,

and, being at a loss as to what to do, had selected medicine on the advice or at the suggestion of relatives or friends, who pointed out to them that the profession was a respectable one which might be expected to yield a reasonable competence. The proportion who stated that they had commenced the study of medicine because of a strong personal inclination or desire would, I am inclined to think, probably be small.

Modern psychology has emphasized the influence of impressions received in early life upon the future of the individual. A chance word may leave a permanent mark for good or ill.

Some years ago I happened to be sitting at dinner next to one of the leading surgeons of the day. "Why was it," I said to him, "that you became a doctor?" "It is interesting," he replied, "that you should ask me this, for only the other day my wife put the same question to me, and I will tell you how it came about. None of my people had been doctors, and I had no bias towards medicine, but I made up my mind to be a surgeon one day when I was only six. My people had a country house and it was on a beautiful summer afternoon that I was lying on the grass beside the drive, cutting up a dead field mouse with a rusty pair of scissors—a thing that any boy might do—when my father and mother came strolling along arm in arm; he, I remember, was smoking a cigar; I can picture the scene as if it had happened yesterday. They stopped some yards away, and my father said to my mother: 'That boy is going to be a surgeon.' My mind was made up, and from that moment I never wavered."

The business attitude of mind may show itself at a very early age.

It was at a private luncheon many years ago that, to my embarrassment, my host called out from the other end of the table: "You know it was your father who was responsible for my entering the medical profession. When I was a small boy he came to see a relative of mine, and after he had left, I heard my mother say to my father: 'Did you remember to give the doctor his cheque?' 'Well,' I said to myself, 'if doctors earn cheques so easily that's the profession for me.' But", he added with a chortle, "since then I have realized my mistake."

Commercialism in medicine is to be deplored. We pride ourselves that ours is a profession and not a trade.

Should a boy desire to adopt medicine as a career he need not be discouraged, as he is apt to be, because he happens to have a "slow brain" or a defective verbal memory, and is in consequence but an indifferent examinee. Individuals vary greatly in the readiness with which they absorb information which is conveyed by the spoken or printed word. Two of the finest clinical observers of my acquaintance—men who made world-wide reputations—had very defective verbal memories, and some of our greatest scientists have been handicapped by the same defect. The faculty of acquiring information rapidly may indeed be a positive disadvantage, for it is a well-known fact that information which is rapidly absorbed is often but poorly retained. A retentive verbal memory is a great asset, but I would remind you of John Buchan's words: "The purpose of education is to train the mind and not to crowd the memory." Information must be digested and form appropriate relationships by association with preexisting knowledge to be of real value; a man who is a veritable encyclopædia

of information may be but a poor thinker and devoid of all originality. He who would be a good clinician must cultivate the attitude of mind of the naturalist with man as his life's study.

Accurate observation and correct inference are here essential attributes. The clinician who is gifted with scientific curiosity and would add something to the sum of knowledge must allow new observations which arouse his interest to "simmer in the mind". Thus he develops a particular type of memory adapted to his purpose, for individual observations magnetized by interest act like nuclei in that they attract information which has a cogent bearing.

As one enters the Royal College of Physicians of Edinburgh, life-size portraits of two former presidents meet the eye. On the right hangs a portrait of Sir James Simpson, the introducer of chloroform, by the Scottish artist MacBeth; on the left a portrait of Dr. Alexander Wood, the inventor of the hypodermic needle, painted by Sir Watson Gordon, Raeburn's most distinguished pupil. The walls of the handsome entrance hall are otherwise devoid of decoration. Possibly the architectural critic may say that these portraits tend to detract from the general effect produced by the simplicity of design, perfect proportions and quiet dignity of the grand staircase which leads up to the College Hall, but, in my opinion, this is more than compensated for by what the portraits symbolize—two of the great ideals of medicine—the relief of suffering and the promotion of progress.

It is of progress I would speak. After reviewing the most striking recent advances in our knowledge of diseases of the nervous system, the late Dr. James Collier concluded his brilliant Harveian Oration of last year with the words: "Neurology has made a good beginning." This is an attitude of mind I would commend to all who are responsible for the training of the undergraduate: medicine has made a good beginning. After reading an exhaustive monograph one may flatter oneself that one knows all about the subject with which it deals. True one may know all that is known, but this may be a very different thing from knowing all there is to know. While the teacher of medicine must always be as dogmatic as the facts permit, he should at the same time try to be suggestive in his teaching, for there are perhaps some among his pupils, he never knows, who if imbued with the spirit of scientific curiosity, which it is in his power to create, may in days to come add to the store of knowledge, and even put corner stones into the edifice of medicine. The premature death of John Hunter, a graduate of whom Sydney may well be proud, whose attainments and accomplishments gave promise of a career which, had he been spared, might have rivalled that of his illustrious namesake, was a calamity to medicine. He has left us an example which will live and go down in the annals of history to the glory of his school.

The man who walks through life without imagination is to be pitied. I would compare him

to one whose delight it is to travel along the main thoroughfares in a high power motor car and who has little inclination to look to either side; he has not learned the pleasure there is in exploring the country lanes, he has not experienced the joys which the countryside affords. When one is young it is the novel and unusual which appeal, but as the years pass—this at least has been my personal experience—the commonplace assumes a quite unexpected fascination, and the limitations of one's personal knowledge and of knowledge in general continue to impress. There are, however, exceptions to every rule, and the exceptional may assume a peculiar interest on account of the light it often throws upon the commonplace. I admit that I am wandering, but thoughts such as these stimulate the imagination, and the unknown gives to medicine an added charm.

Before I proceed to cite some illustrations of methods which have been responsible for progress, I would correct a very prevalent misconception as to the meaning of the term research. The word should not be restricted in its usage to the methods employed and the results obtained by the laboratory worker, but should be used to include all inquiry the purpose of which is to ascertain the truth; clinical investigations, carried out in the hospital on scientific lines, are just as deserving of the epithet "research" as are the pursuits of him who works in the laboratory with the microscope, the test tube or experimental animal. The physician who assays to prove the effect of a drug at the bedside in cases of disease, employing true scientific methods for the purpose, deserves the title of research worker just as surely as does the pharmacologist who arrives at his conclusion from experimental observations in the laboratory. Nay more, I would affirm that the difficulties of drawing conclusions at the bedside are much greater than those that confront the laboratory worker.

The advances in medicine have been of various kinds, and have been achieved by different methods of approach, to some of which it is my present purpose to refer.

Hypotheses, which to us appear absurd, dominated medical practice in our forbears' time. As an extreme instance of grotesque reasoning, I recall the treatment of shingles, a cure regarded as infallible, which was practised in China up to quite recent times, and may be at the present day for all I know. The Chinese physicians were struck by the resemblance of the characteristic groups of spots, the so-called herpetic vesicles, to berries. They painted a plate around the vesicles and beside the plate a bird with open bill. When the vesicles disappeared, as they invariably do, it was believed that they had been eaten by the bird. The *post hoc* assumption is a common source of fallacy.

Accurate observation and sound inference are essential for all progress. Sydenham it was who laid the foundations of medicine as a science, when, discarding tradition and hypothesis, he went directly to the patient to make his observations and

drew conclusions from the facts observed. He was indeed the father of clinical medicine. A rich harvest of information, as to the nature and diagnosis of disease, has accrued from the study of the symptoms presented by the patient during life and their correlation with the changes observed in the organs after death.

Experimental medicine may be said to have been born when William Harvey, often referred to as the father of this method of research, discovered the circulation of the blood. In his work "*De Motu Cordis*", one of the world's greatest literary treasures, a masterpiece of accurate observation and scientific reasoning, Harvey exhorts his readers to put their theories to experimental proof.

Two discoveries, two of the greatest discoveries of all time in relation to the healing art, the introduction of antiseptics by Lord Lister and of chloroform by Sir James Simpson, were of inestimable value in opening up the possibilities of the experimental field. We in Edinburgh are justly proud of these two pioneers, who held the Chairs of Clinical Surgery and Obstetrics in our University.

The name of Sir Charles Bell, another alumnus of our school, where he was the Professor of Surgery just over a hundred years ago, comes to my mind. Bell discovered by experiment the functions of the anterior and posterior spinal roots, and formulated a conception of the nervous system which in its main features stands today. Indeed, I ventured to suggest in my Harveian Oration at Edinburgh last year that Bell might be described as the discoverer of the circulation of nerve impulses just as Harvey is credited with the discovery of the circulation of the blood. Bell was a sensitive man who loved animals and disliked experiment; had he lived after the discovery of anaesthetics and antiseptics, one wonders how far his genius would have carried him.

Experiment has indeed done much to expedite progress. A few days ago, in opening a discussion at Melbourne upon tumours of the brain, I reminded my audience that it is just fifty years since the surgeon first removed a tumour from within the skull. The precise localization of the tumour was made possible by the experimental work of Fritsch and Hitzig, and of David Ferrier, for it was these observers who demonstrated to the world that particular functions are located in particular portions of the brain.

Discoveries may be arrived at, so to speak, by accident. A striking case in point is afforded by the discovery of the cause of myxoedema. This disease, which is most commonly met with in women near the change of life, is characterized as you know in its extreme forms by a waxy-like skin, increase in weight, absence of perspiration, falling out of hair, slowing of the pulse, a subnormal temperature and a variety of other symptoms. For years the cause of myxoedema remained unknown; it may be said to have been discovered by chance. An enlargement of the thyroid gland, known as a

simple goitre, is very prevalent in the Swiss Cantons. These goitres often attain a large size; their appearance is unsightly, and they may cause breathlessness by pressure upon the windpipe. The Swiss surgeons are often called upon to operate and remove the tumour, and it was Kocher, the distinguished surgeon of Berne, who noticed that when the whole thyroid gland was removed these patients subsequently developed the symptoms of myxoedema. That myxoedema must be due to loss of function of the thyroid gland was an obvious inference. Victor Horsley, brilliant alike as physiologist and surgeon, was quick to grasp the practical application of this observation. He proved that the symptoms of myxoedema appeared in the sheep after removal of the thyroid gland, and that the transplantation of a healthy gland into the myxoedematous sheep resulted in temporary benefit. The next step was made by George Murray, who succeeded in producing the symptoms of myxoedema in rabbits by thyroidectomy, and then cured the condition by repeated injections of extract of the gland. Most of us have probably had what we regarded as bright ideas from time to time, and have regretted that we have not followed them up. I remember during my students days—I was attending physiology at the time—discussing Murray's discovery with a friend, and suggesting to him that since the presence of the *foramen caecum*, or blind opening, near the root of the tongue would appear to indicate that at one period in the process of evolution the thyroid gland almost certainly poured its secretion directly into the alimentary canal, it might be worth while trying the effect of giving thyroid gland by the mouth to patients suffering from myxoedema. It was soon after this that Sir Hector MacKenzie proved the efficacy of oral administration. We know that the symptoms of myxoedema are due to absence of the thyroid secretion, that they disappear completely when the patient is fed on thyroid, and that he will remain free from the symptoms of the disease so long as the thyroid is continued. On the other hand, we are quite ignorant as to why it is that the thyroid gland atrophies or withers and thus determines myxoedema.

Endocrinology, the term applied by Sir Edward Sharpey-Schafer to the ductless glands and their diseases, has made great advances of recent years. Sir Edward, the doyen of physiology, held the Chair of the Institutes of Medicine in Edinburgh for over thirty years. During his lifetime a lectureship was founded in his honour—a rare distinction. He died early this summer, a few weeks before Sir Charles Sherrington delivered the Sharpey-Schafer Lecture in which the lecturer described in graphic language the profound impression created when Schafer and Oliver demonstrated, at a meeting of the Physiological Society in 1894, the remarkable effect of the experimental injection of suprarenal extract. All who were present, said Sir Charles, realized that they had witnessed an epoch-making discovery, an experiment which opened up unlimited

possibilities for research and marked indeed the birth of a new science.

The action of the nitrites in relieving the terrible pain in many cases of *angina pectoris*, a discovery which has been of inestimable value in the relief of suffering, was demonstrated by Lauder Brunton while he was a house physician in the Royal Infirmary, Edinburgh. Impressed by the hard contracted radial artery of a patient under his care during attacks of *angina pectoris*, and by the flushing of the skin which his friend Gamgee had shown him was produced by the nitrites with which he was working in the laboratory, the idea occurred to Brunton that the pain of *angina pectoris* might be due to the contracted state of the arteries, and that if the nitrites dilated the arteries, as they did the capillaries, the pain might thereby be relieved. And his reasoning proved to be correct.

Banting's discovery of the insulin treatment of diabetes provides another illustration of what may be achieved by one who has had no special experience of research. After reading one evening an article in *The British Journal of Surgery and Gynaecology*, he wakened in the early hours with his inspiration. Sleep was no longer possible, for he was obsessed with the urge to put his idea to experimental proof; he acquired the animals necessary for his experiment and proved his point. It is strange, is it not, that when many brilliant investigators had been for years engaged in research with a view to finding a cure for diabetes, one who had had, I think I am right in saying, no previous laboratory training, should make this great discovery?

Some of the most important advances in medicine, especially in relation to diagnosis, have been directly due to the introduction of instruments of precision.

The stethoscope was invented by Laennec. Auscultation had been practised previously, but the method of examination was thereby simplified and attention was in consequence concentrated upon the cardiac and respiratory sounds. Laennec's invention and his work upon auscultation made Paris the Mecca of medicine a century ago.

James Mackenzie, an Edinburgh graduate and a general practitioner, when sitting by the bedside of women in labour, found the study of the pulse a pleasing recreation. The polygraph which he devised, by means of which simultaneous records of the pulse, the heart beat and the respirations could be obtained, opened up the mechanical study of cardiology. The use Mackenzie made of his invention gave to this department of medicine the greatest impetus it had received since the days of William Harvey.

Blood pressure is nowadays a byword with the public. The introduction of the sphygmometer, the instrument by which the blood pressure is estimated, may be described as a mixed blessing. The practitioner, it is true, is now possessed of a simple and impressive procedure, the patient flatters himself that he has now something he can understand, and

the actuary, who deals with life insurance, is delighted to find something in medicine which can be expressed in precise figures. But while the sphygmometer is a valuable instrument when the information it affords is assized by a competent clinician who recognizes that the readings he obtains must be correlated with the other data which the case presents, and who possesses the faculty of seeing a case from the patient's point of view, it is a dangerous weapon in the hands of a medical man who glibly talks of reducing a raised blood pressure as his essential aim, quite irrespective of the cause, and who acquaints the patient with his readings, for much unnecessary anxiety is often so produced.

I have referred at random to instances as they occurred to me which illustrate some of the methods by which advances in clinical medicine have been achieved. Would that I had time to tell you more of the history of the progress of medicine; of the knowledge afforded by the microscope as to the minute structure of the organs of the body, the blood and the various fluids in health and disease; of the science of bacteriology and the wealth of information which has accumulated since Koch discovered the bacillus which is the cause of tuberculosis, and of the advances in chemistry, and the rich field of promise which has thereby been opened up. These are but some of the many lines along which medicine has advanced and is advancing.

The incidence of disease, I would remind you, is now very different from what it was. Preventive medicine has made great strides. As a consequence of the improved standard of living and administrative control, cholera, typhus fever and relapsing fever have disappeared; vaccination has practically exterminated smallpox, and has been of inestimable value, as the experience of the Great War proved, in the prevention of enteric fever; the recognition of the causes of beri-beri, malaria, yellow fever and sleeping sickness has resulted in the introduction of preventive measures which have very materially diminished the incidence of these disorders; and coordinated campaigns directed against tuberculosis and syphilis, in conjunction with the new remedies utilized in the treatment of the latter disease, have had a profound influence upon the morbidity of these scourges. At the present day chlorosis, the anæmia of young girls, at one time so common, is a rarity; scurvy is very seldom seen; acute gout is very much less frequent than it used to be; and rickets is disappearing; while pernicious anæmia and diabetes, two disease which a few years ago were almost invariably fatal, can now be effectively dealt with. But while progress has been great, there are still many diseases which are as prevalent as ever they were. Cancer and the rheumatic disorders still defy us, and no measures are yet known which curtail the spread and influence the course of influenza and "the new disease", lethargic encephalitis or sleepy sickness.

But there is a personal side to medicine to which it is only right I should refer, for the manifesta-

tions or symptoms of disease may be due to mental as well as physical causes, and treatment may be mental as well as physical in its mode of action. Many of the headaches, neuralgias, dyspepsias and a host of other disorders met with in practice are not due, as the patient often thinks, to physical disturbances, but are a direct consequence of anxiety and mental misconceptions. To these disturbances of mental origin, which are quite distinct from the insanities and psychoses, the term neurosis is applied.

The neuroses have of late been the subject of much attention. It was Freud who created a new interest in this department when he advanced the view that these disorders are commonly determined by long forgotten impressions received in early childhood, and at the same time devised the method of treatment known as psychoanalysis by which the causative complexes, as they are termed, are brought into consciousness and their ill-effects thereby neutralized. My special purpose in mentioning psychoanalysis is to correct an unfortunate misconception which is very prevalent. Psychotherapy and psychoanalysis are not synonymous terms. By psychotherapy we mean all those mental influences and methods of treatment, and they are various, which are employed to benefit the mental factor in disease. Psychoanalysis, on the other hand, is a very specialized procedure which is utilized to explore the unconscious mind. Psychotherapy in one form or another is constantly called for in the treatment of the neuroses and the mental factor in illness; but psychoanalysis, a dangerous therapeutic measure in unskilled hands, which has been and still is much abused, is applicable only to a very small proportion of specially selected cases in which other methods of treatment have failed.

A reference to what may be called pseudo-scientific medicine may not be out of place. As Oliver Wendell Holmes once remarked with reference to medicine, even a man of exceptional ability may be quite at sea in connexion with matters which lie outside his line of thought. The public is extraordinarily gullible. A man who is ill is so easily taken in by pseudo-scientific explanations and by advertisements extolling the virtues of quack remedies in which he sees his own symptoms so cleverly depicted. Fortunes have been made by the sale of proprietary remedies. It is only right that the layman should know that these remedies have been analysed and reported on, and that almost without exception they have been found to contain either the same ingredients which the medical man is constantly employing in the ailments which they profess to cure, or innocuous substances which have no influence upon disease apart from the hope and faith inspired by their name. We medical men make no claim to be cure-all agents; we pride ourselves that we are students of disease whose purpose it is to study disease in all its aspects and try to ascertain the truth.

In conclusion, I would express once more my appreciation of the honourable position in which I

find myself this evening, and the pleasure it has given me to visit your beautiful capital. May all good fortune attend this magnificent institute, the material and intellectual foundations of which have been so surely laid by Sir Colin MacKenzie.

#### THE CLINICAL RECOGNITION OF THE ARRHYTHMIAS AND THEIR TREATMENT.<sup>1</sup>

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In this article it is my aim to discuss the recognition of the arrhythmias from a purely clinical point of view, on the assumption that instrumental methods of recording disorders of the heart are not readily available. Indeed in most cases graphic records can be dispensed with, though their value is unquestioned in diagnosis, in research and for future reference.

I shall avoid a full discussion of theory and the rarities will be largely ignored so as to make the paper of practical use.

The normal beat of the heart arises in the pacemaker area, known as the sino-auricular node, at the junction of the superior *vena cava* and the right auricle. It is richly innervated by the vagus and sympathetic, and is readily influenced by nervous mechanisms. Impulses generated here sweep through the auricular musculature till they reach the auriculo-ventricular node.

The auriculo-ventricular node is part of the bundle of His, and the excitation passes down this conduction neuro-muscular track in less than 0.2 second. Thence it traverses the two main branches proceeding to the left and right ventricles and rapidly spreads out into the ventricular muscle by way of the Purkinje network. Contractions arising from impulses other than the normal give rise to extrasystoles.

#### Sinus Arrhythmia.

The physiological arrhythmia known as sinus arrhythmia will be discussed first. It is quite harmless, but may be a source of confusion in diagnosis, so that its recognition is essential. It is due to variations in vagal tone, usually in association with the respiratory cycle. The heart slows in expiration and quickens in inspiration. In older people it may occur independently. It is very common in young children and adolescents and is constant in the dog.

It is most likely to be mistaken for serious irregularity when it is noted in the convalescent stage of diphtheria and rheumatic fever or in toxic hearts after tonsillitis and influenza.

<sup>1</sup> Read at a meeting of the Victorian Branch of the British Medical Association on November 23, 1935.

It may raise the question of normality when found in routine examination of young people for civil service appointments, or in the examination of school-boys, especially when a heart bruit is also discovered, or after complaint of precordial pain or faintness. Individuals may have their future jeopardized when functional bruits and sinus irregularities are mistaken for something serious.

This irregularity may be found when increased intracranial pressure is present, as in tuberculous meningitis and in head injuries and tumours. It is this unhappy association which in the past has given this rhythm an evil undeserved reputation.

Sometimes sinus irregularities take the form of long pauses and may be mistaken for heart block. The points to recognize are: (i) that the heart sounds are normal; (ii) that the force of the pulse beats is equal; (iii) that the irregularity consists in a waxing and waning of the heart and pulse rate, it slows in expiration and quickens in inspiration; (iv) the irregularity may be made more evident by forced respiration; (v) quickening the heart rate abolishes the irregularity. This may be effected by emotion, exercise, or by drugs, such as atropine and amyl nitrite.

The arrhythmia is not found whilst the rate is rapid, whether this results from fever or from other causes. It may be quite marked during convalescence and usually it is then that doubt arises regarding interpretation. The above tests usually serve to distinguish it; one must remember that it may not be safe to test a convalescent diphtheritic heart by exercise. Sometimes a mother discovers the irregularity by hearing the heart whilst lying next to the patient.

Treatment is unnecessary. The main thing is reassurance that it is quite harmless and to request the anxious parent not to take the pulse and not to create apprehension and anxiety. It is quite easy to manufacture cardiac neurosis by injudicious management or by unguarded statements.

#### Extrasystoles.

Perhaps the most common of all the irregularities of the heart rhythm are those due to extrasystoles or premature beats. They may appear at any age, but are certainly more common after middle age and are infrequent in childhood.

They are beats which arise from some focus in the heart muscle other than the usual area for impulse formation. Ectopic impulses which arise outside the normal area give rise to premature contractions that interrupt the normal rhythm. The new site of impulse formation may be in the auricle or in either ventricle or in the region of the auriculo-ventricular node. The clinical recognition is usually easy, and often extrasystoles can be diagnosed with surety even from the patient's description of the sensation caused by the intermittent heart action.

When asked to describe the character of the palpitation, patients make use of such expressions as "the heart stumbles" or "stops" or "thumps" or "rolls over". Any description implying an isolated intermittent beat or a pause or an occasional

thumping sensation over the heart area can be safely regarded as due to extrasystoles. An apt comparison is to a motor car engine missing fire in one cylinder.

These premature beats may occur at any time and in any frequency. They may be quite infrequent or, on the contrary, may occur often. Sometimes they are arranged in regular order, as when each normal beat is regularly followed by an extrasystole and a pause. This form of grouped beats is known as coupled beats, and the heart may continue to beat in this order for any length of time, ranging from a brief period to hours, days or weeks. I have instances in which it has continued for many months. In one case this rhythm persisted unchanged for two hours all through an operation for acute mastoiditis, under intratracheal ether anaesthesia. The circulation was not embarrassed by the coupled beats, due in this instance to auricular extrasystoles.

The contraction of the ventricle which results from the premature impulse may or may not expel sufficient blood to open the aortic valves. If it does, a small premature beat may usually be palpable at the pulse; if not, a pause or dropped beat is noted. With auscultation, recognition of extrasystoles is usually easy. Corresponding with the premature contraction, one hears two little sounds if the aortic valves operate, or only one little sound if the valves do not open. If this is so, a complete intermission of the pulse is noted. In these cases the pulse rate is half that of the ventricular, usually at about 34 to 40 per minute. Such slow pulse rates may be mistaken for heart block. But such an error can be easily avoided by noting the arrangement of the heart sounds. The first and second normal heart sounds will be linked to a little sound, then follows a pause, and again in turn appear the two normal sounds and the single sound for the extrasystole, and so on. With exercise it is often possible to cause disappearance of the coupling, at least for a time.

When only a single sound is heard corresponding to the premature beat, the origin is in the ventricle, because with auricular extrasystoles enough blood is sent down to the ventricle to enable the ventricle to raise sufficient pressure to open the aortic valves, and therefore two sounds will be heard.

Coupled beats which result from excessive dosage of digitalis in cases of auricular fibrillation show similar signs and may or may not be associated with halved pulse rates. The chief difference between coupled beats associated with auricular fibrillation and those associated with normal rhythm is that in the former the pauses are uneven. When a premature beat is regularly felt at the wrist following a normal beat, it is known as *pulsus bigeminus*, and when two normal beats are regularly followed by an extrasystolic beat we have *pulsus trigeminus*.

Extrasystoles usually give rise to difficulty in diagnosis only when they occur very frequently or from multiple foci. The rhythm then simulates auricular fibrillation. Frequently it is possible to differentiate by causing the heart rate to quicken,

when, as a rule, the heart becomes more regular; in fibrillation the rhythm becomes more irregular as the rate increases. Extrasystoles associated with gallop rhythm create a confused medley of sounds which also simulate auricular fibrillation. Here, by observing that the pulse is regular, except for the intermittent pause or premature beat, one can exclude fibrillation.

Whilst it is generally true that extrasystoles usually appear only when the heart rate is not rapid, this should not be stressed over much, as extrasystoles do appear in some hearts with rates in the vicinity of 120 per minute. Usually this indicates excessive myocardial irritability. Also, whilst it is usual for extrasystoles to disappear with quickened rates, this is by no means invariable, and in not a few cases of grossly irritable myocardium exercise may actually provoke extrasystoles.

Such cases tend to paroxysmal tachycardia and to auricular fibrillation. This is well illustrated in cases of advanced mitral disease, toxic goitre and other toxic heart conditions.

It is a frequent experience for extrasystoles to interrupt the normal rhythm when the heart slows. For this reason extrasystoles frequently appear when the patient is dropping off to sleep, and the unpleasant sensation of the heart stopping or thumping may wake the patient with a start and create much fright in nervous subjects.

The anxiety syndrome often includes faintness, pallor, sweating, tremors and apprehension of death, and may be mistaken for cardiac collapse.

Another sign sometimes produced by extrasystoles is a sudden jerk visible in the jugular veins. This occurs when the extrasystole causes the auricles and ventricles to beat simultaneously; the inflow of the venous blood is abruptly checked, and also a pulsation is abruptly transmitted to the vessels in the neck. The sudden throb may cause an unpleasant sensation and often a feeling of chokiness.

How are extrasystoles caused? This is not precisely known. The premature beat is produced by some form of sudden irritation in the heart muscle. It may be a chemical or a physico-chemical effect, or a nervous excitation. There is evidence to show that vagal and sympathetic effects are produced by the formation of chemical substances which accumulate rapidly and are as rapidly broken down. The vagal substance is similar to acetyl choline, and the sympathetic to adrenaline.

The premature beat, contrasted with a normal beat, is characterized by the rapidity with which the impulse is built up. It is for this reason that it is premature, and also that the ectopic beat is less usual when the heart rate is hurried, as there is less opportunity for the premature impulse to cut in. From a clinical point of view, the exciting causes may be grouped as reflex, toxic, nutritional and nervous. Reflexly they may occur from the effects of a full meal or from air swallowing. A

good belch, if not good form, is nevertheless effective. Reflex excitation is a very common association with flatulent indigestion and temporary distension of the stomach. Lesions of the stomach, duodenum and gall-bladder readily cause reflex disturbance of the heart, no doubt by reason of the vagal and sympathetic innervation common to these organs. Such reflex stimuli conceivably may liberate rapidly a chemical excitant to the heart muscle.

Concerning toxic causes, cigarette smoking is a frequent offender. Chronic focal infection may also be operative. Sometimes also acute infections; and digitalis may induce extrasystoles by irritating the heart muscle. Coronary sclerosis and occlusion often cause extrasystoles. Emotion may move the heart tumultuously by indirect action, acting through the vasomotor and endocrine mechanisms, and extrasystoles may occur.

#### *Treatment of Extrasystoles.*

It must be confessed that there is no treatment guaranteed to suppress frequent extrasystoles. Usually patients can be reassured that this form of palpitation is more of a nuisance than a disease and should, as far as possible, be shut out of conscious perception and ignored. Obviously ætiological factors should be searched for and, if possible, eliminated. Smoking and the taking of alcohol may need to be discontinued. Focal infection should be attended to, but one's experience here is that there are "many losers and few winners". We are inclined to remember the successful cases and to forget about the others, or the patients forget us and go for relief elsewhere.

Of drugs, sedatives are the most useful, either in the form of bromide or phenobarbitone. These are especially valuable for those patients who are disturbed by extrasystoles at bed time. Ammonium bromide, in doses of 1.2 grammes (20 grains), or phenobarbitone, 0.03 to 0.06 gramme (half to one grain), usually dampens down the perception of the intermittent action of the heart and allays nervousness. Wenkebach advocates small doses of digitalis. I have not found it useful; and the same may be said of small doses of strychnine, quinine and atropine. Quinine hydrobromide, in 0.18 to 0.3 gramme (three to five grain) doses, is more serviceable. Quinidine sulphate, 0.09 to 0.18 gramme (one and a half to three grains), taken three times a day, is more successful, but it is not well tolerated by all patients. It may induce looseness of bowels, nausea and faintness.

Alimentary disorders should be corrected by attention to diet, by unhurried mastication, and by the use of gastric sedatives and carminatives. Removal of a diseased gall-bladder is often effective in removing this and other forms of heart disorder.

If the heart is otherwise sound, no restrictions need be imposed as regards exertion or vocation, and anaesthetics may be administered without fear.

### Paroxysmal Tachycardia.

Paroxysmal tachycardia is a relatively common disorder, if one includes cases based on a characteristic history and not merely those actually observed during an attack. Frequently it has ceased before the arrival of the medical attendant.

Usually the patient will graphically describe a sudden onset of the rapid action of the heart, as if it raced or fluttered. Ask for the rate and action to be tapped out by the finger, and usually the extremely quick beat can be easily identified as due to paroxysmal tachycardia. The abrupt onset is very typical; it comes like a bolt from the blue; the heart races for a time, then ceases just as abruptly, ending with a thump; then quiet action follows, much to the relief of the victim. Attacks may occur in any frequency and for most varied durations. Some patients may have attacks only at very long intervals, and others have attacks every few days. The attack may last for a few seconds, minutes, hours or even days. Fortunately, the longer attacks are less usual. I have records of one case in a lad of twenty, in whom a paroxysm lasted for twenty-one days with survival. Unfortunately, in the next attack he developed acute pulmonary oedema and died.

The attacks are often preceded and succeeded by extrasystoles; the form of these, as shown in the electrocardiograms, may disclose the origin and nature of the paroxysm. The tachycardia may originate in the auricle, in either ventricle, or at the auriculo-ventricular node, but this is mainly a matter for electrocardiographic study, and this paper is not concerned with this aspect of the subject.

Paroxysmal tachycardia includes not only these regular paroxysms, but also paroxysmal attacks of auricular flutter and fibrillation.

The attacks may occur at any age from infancy to senility. I have records secured from two infants, one at the age of nine weeks, who has survived and who is now a healthy child of five years. The rate in this case was recorded by the electrocardiograph at 300 per minute. The other patient, an infant of seven months, succumbed to the condition. At the other extreme I have seen cases among patients in the eighties. However, the usual age is from early adult life onwards. The attacks may occur in association with mitral and aortic disease, coronary sclerosis, hypertensive heart, goitre heart, and other forms of organic lesions. On the other hand, a considerable number of affected persons show no signs whatever of cardio-vascular lesions. The exciting causes are similar to those noted in extrasystoles. Some quite trivial incident may start a paroxysm, such as stooping. Sometimes a paroxysm comes on while the patient is at rest, sometimes with exertion or excitement.

The degree of symptoms varies greatly. Some patients are entirely unconcerned and others extremely apprehensive. Symptoms are governed by the following factors:

1. The heart chamber in which the paroxysms originate. More distress occurs from ventricular paroxysms than auricular, whilst nodal types are intermediate in severity.

2. The state of the myocardium. An otherwise healthy muscle stands up to the burden without failure, whilst with myocardial degeneration a dangerous state may ensue.

3. The duration of the paroxysm. Attacks which last many hours may bring on a serious train of symptoms, but here again this depends on the underlying integrity of the myocardium.

4. The temperament of the patient is a big factor. In hypersensitive neurotic patients alarm and apprehension are great and may be associated with collapse, whilst phlegmatic persons may be extraordinarily unconcerned.

5. The attitude of the physician is of some importance in this regard. A demeanour calm and unruffled, but devoid of casualness, will convey confidence, whilst a fussy, nervous management will surely add to the patient's alarm. Sir William Osler emphasized the value of equanimity and imperturbability. These qualities should be applied in this, as in all states of emergency.

6. Another factor in symptoms is the build of the patient. A stocky person with a roomy chest is much less likely to be conscious of his heart action than a long, thin, hypotonic, asthenic individual, whose heart knocks unpleasantly at his bony thorax.

In most cases the palpitation and racing action are not the only complaints, but there is also an unpleasant throbbing in the neck, which, by beating against the trachea, may evoke suffocating and choky sensations.

On examination the pulse rate is very rapid. Usually it is about 180 per minute, but rates range from 120 to 240. The lower speeds would be more difficult to identify than the higher rates. In paroxysmal fibrillation the ventricular rate is about 130 to 180 per minute. It may be difficult to count the pulse, especially when *pulsus alternans* occurs. The heart sounds are weak and tic-tac, and the blood pressure falls. The veins of the neck become distended and the heart may progressively dilate. The liver may become engorged, and crepitations may be heard in the base of the lungs. Oedema of the legs is not usual. Precordial pain may be complained of. However, in many cases it is remarkable how few signs of congestive failure appear. When the attack ceases, improvement is dramatic; the signs of dilatation and congestion disappear and the patient feels comfortable, though maybe exhausted and tired.

For the clinical recognition of paroxysmal tachycardia attention should specially be paid to: (i) the mode of onset and offset (abruptness is very characteristic, particularly of the onset); (ii) the maintenance of the rate at an even speed. The rate is not slowed by rest nor quickened by mild exertion, as is the case with simple tachycardia. This does not apply to paroxysmal fibrillation, where the ventricle does quicken with movement.

*Treatment of Paroxysmal Tachycardia.*

We can occasionally learn clinical tips of value from patients, and this applies to the management of paroxysmal tachycardia. In the first place try the effect of simple procedures which have formerly proved successful. Ask the patient to take and hold a very full breath for as long as possible. I have seen this arrest attacks instantly. Try the effect of pressure on the neck over the right carotid sinus. This should be maintained for about twenty seconds. Next induce retching by tickling the pharynx, or after the swallowing of two large glasses of warm solution of sodium bicarbonate. Sometimes eructations of gas from the stomach succeeds, and this may be assisted by the use of alcohol or carminatives such as syrup of ginger, peppermint or aromatic ammonia.

The act of swallowing a capsule arrested one attack. Vaquez mentions a similar instance. It is likely that the act of retching or swallowing induces vagal inhibition. Certain drugs, by producing vomiting, may thus be incorrectly credited with success. This sometimes happens after administration of morphine.

Any or all of these procedures may fail. Always remember that the tendency is for the attack to stop spontaneously. This makes one chary of ascribing success to a particular drug or procedure, but it is a good reason why we should adopt a hopeful attitude. Remember also that a procedure successful on one occasion may fail at another time.

After trying the above simple methods without success one has recourse to drugs. I usually prescribe one large dose of digitalis and wait one to two hours. Two cubic centimetres (one drachm) of tincture may be given, or three tablets of digoxin or three white Nativelle's granules (equivalent to 0.8 milligramme or one-eightieth of a grain). There are many similar preparations which need not be enumerated. If unsuccessful, then give quinidine sulphate, 0.18 gramme (three grains), each hour for four doses; if necessary, repeat the digitalis and then the quinidine. Sometimes larger doses of quinidine are used if the drug is well tolerated. In more desperate cases I have succeeded with intravenous injections of quinine bihydrochloride, 0.3 gramme (five grains), but have also failed with this dose and with 0.6 gramme.

Occasionally I have tried intravenous injections of quinidine sulphate in 0.18 to 0.3 gramme (three to five grain) doses, but it is by no means uniformly successful. I have tried "Solvochin" (25% solution of quinine) in one case only and failed to produce any effect, except phlebitis for several inches in the brachial vein. The patient in this instance was a man aged sixty years, who had very persistent tachycardia at 180 per minute following coronary thrombosis. He died a week later.

One of my patients, whose paroxysms occur at 140 per minute, frequently arrests them by taking a trinitrin tablet (0.65 milligramme or one one-hundredth of a grain). Many other drugs have

been tried, but with most uncertain results. Quinidine is the most reliable remedy, but it is not devoid of risk. In common with other observers, I have entirely failed to influence some paroxysms, but fortunately the majority end spontaneously, even without treatment.

*Auricular Flutter.*

Little time need be given to auricular flutter. It is a rare disorder and usually requires graphic records for its identification.

Briefly, it consists in a rapid rhythm in the auricle at about 240 to 300 per minute. The excitation waves follow a circuitous pathway round the orifices of the *vena cava*. Impulses reach the auriculo-ventricular node regularly, but as a rule only half the number are conducted to the ventricle. In this sense the condition is an auricular tachycardia associated with 2:1 heart block. Sometimes the ratio is 3:1 or 4:1, or the ratio may vary frequently.

The clinical indications of its presence are: (i) a persistent rapid heart rate in elderly people not otherwise explained; (ii) a rapid fluttering pulse in the neck veins; (iii) a sudden doubling of the heart rate, for example, 120 to 240 per minute when a 1:1 ratio occurs; (iv) vagal pressure may markedly slow or even arrest the ventricles.

Some prolonged attacks of unconsciousness are due to ventricular tachycardia, when all the auricular impulses pass to the ventricles at very high speeds, such as 280 per minute. The output and blood pressure are very low, with consequent cerebral anæmia.

Attacks of flutter may be paroxysmal or they may continue for years.

*Treatment of Auricular Flutter.*

Digitalis usually slows the tachycardia, and often it converts it into auricular fibrillation. When this happens, withdrawal of the drug may be followed by restoration of normal rhythm. Quinidine slows the auricular oscillations, but is less successful than it is in fibrillation for restoring normal rhythm.

*Auricular Fibrillation.*

Auricular fibrillation is perhaps the most important of the arrhythmias, because: (i) it is very common, (ii) it is a frequent cause of rapid heart failure, (iii) it usually responds excellently to adequate treatment, (iv) its clinical recognition is not difficult.

The mechanism of this irregularity may briefly be referred to. It is due to replacement of the normal sino-auricular nodal impulses by a rapidly circulating wave around a shorter path than in flutter. Electrocardiograms show that the oscillations are about 450 per minute. At this high speed the auricular fibres have not time to recover from the refractory period evenly. Consequently some fibres are excitable and convey the impulse, some are refractory and block it, some are partially refractory and hinder the wave. The result is that

the oncoming wave of contraction has to follow a sinuous path in the circuit, and therefore impulses reach any particular point at irregular times. The auriculo-ventricular node cannot conduct anything like the huge number of impulses bombarding it from the auricle. A certain number pass along the conduction tissue and activate the ventricles in irregular fashion. The auricle no longer contracts in a coordinated manner, but experimentally flickering fibrillary movements are visible, whilst the ventricular rhythm is completely irregular, both in the time intervals and the force and volume of the beats.

It is this complete irregularity of the heart and pulse which is so characteristic of auricular fibrillation. Its clinical recognition depends on this quality—short pauses and long pauses, large and small beats are mixed indiscriminately. A long pause may follow a large beat or a small beat; there is no orderly arrangement. Increase of the heart rate increases the degree of irregularity—a useful method of distinction.

Frequent extrasystoles from multiple foci simulate fibrillation very closely. So do extrasystoles in association with either sinus arrhythmia, *pulsus alternans* or gallop rhythm. It is also less easy to distinguish auricular fibrillation when the ventricle rate is low than when it is rapid.

Deliberate quickening of the rate will often determine an increase in the degree of irregularity which would argue in favour of fibrillation; conversely, regularity would rule it out.

In cases of mitral stenosis when fibrillation is occurring, the typical rumbling crescendo presystolic bruit disappears. This can best be determined by timing it in the longer pauses. If a diastolic bruit is present before the onset of fibrillation, it will still persist. Auricular fibrillation commonly occurs in the following types of cases: (i) In mitral disease of rheumatic origin. In the terminal months or years fibrillation almost invariably occurs. (ii) In old-standing cases of toxic goitre. (iii) In senile cases. (iv) In cases of arteriosclerosis and coronary sclerosis. (v) In a certain number of cases following coronary occlusion. (vi) A small group occurs in acute infections and also in chronic infection. Curiously, it is very rare in infective endocarditis.

When auricular fibrillation sets in, the pulse rate is usually rapid, but not always. In some cases the bundle of His will not conduct at a high rate and so the ventricle remains relatively slow, although irregular. I have watched two cases of this type for twenty-three years; one patient recently died at the age of seventy and the other is still carrying on.

Again, if the speed of the ventricle is very high, a number of beats fail to reach the wrist—the difference is known as pulse deficit. Fibrillation and heart block may occur together, but this usually demands the electrocardiograph for recognition.

The degree of heart failure varies from case to case, and also the rapidity with which it is induced, but congestive failure and marked engorgement in

the liver and jugular veins frequently occur. When this rhythm occurs, angina is unusual. Paroxysmal auricular fibrillation is not uncommon; frequent attacks coming at short intervals tend to pass into persistent fibrillation.

#### *Treatment of Auricular Fibrillation.*

The key to the treatment of auricular fibrillation is the proper use of a potent preparation of digitalis. Quinidine plays its rôle in a select few only. The details of management of heart failure are so familiar that details regarding rest, position, diet, sedatives *et cetera*, may be omitted, but some consideration must be given to the use of diuretics in heart failure.

In brief, digitalis has three main actions: (i) An indirect vagal effect influencing the sino-auricular and the auriculo-ventricular node. It increases vagal inhibition and thus slows the heart and tends to produce heart block. (ii) A direct effect on the heart muscle independent of vagal action. This is only a small effect. (iii) Some uncertain degree of effect on tone.

In normal rhythm the slowing effect of digitalis is inconsiderable, amounting to six to ten beats per minute. In fibrillation the effect is great, because the degree of heart block induced is quite sufficient to prevent the passage of the feeble impulses generated from the circus movement in the auricle. It is thus easy to slow the ventricle, lessening the work, allowing better filling and a stronger output of blood. A vicious circle is replaced by a favourable one and improvement occurs. Digitalis does not affect the fibrillating auricle. We speak loosely of slowing the fibrillation, really meaning that we slow the ventricle rate by imposing a grade of heart block. Any good preparation of digitalis will do this; the most stable is the powdered digitalis leaf; next is the tincture, and lastly the infusion. The glucosidal principles are active and stable, and rather expensive. The preparations are hydrolysed by water and gradually become inert. For this reason it is not good practice to prescribe digitalis in a mixture. It should be given separately, either as drops with or without other inert flavouring tinctures, or, if desired, as tablets, pills or crystalline digitalin. Hypodermic injections cause irritation and are not well absorbed. For very rapid action intravenous injections may be required occasionally, but here strophanthin is even more potent. Digoxin prepared from *Digitalis lanata* is an excellent preparation when given either by the mouth or intravenously.

The principles of digitalis administration in auricular fibrillation are: (i) to reduce the ventricular rate to 70 to 80 per minute, (ii) to maintain it at this speed indefinitely, (iii) to avoid toxic effects.

If the heart failure is not severe and there is no urgency, give four cubic centimetres (one drachm) of the tincture daily, or 0.18 gramme (three grains) of the powdered leaf or some equivalent dose of other preparation. Continue administration till

the desired effect is obtained or till the toxic effects appear (in about five to seven days), then reduce to a maintenance dose, which is usually 1.2 cubic centimetres (twenty minims) of tincture or a grain of the leaf daily or on alternate days.

The heart must be digitalized for the remainder of life, which may extend for ten years or so. The mistake is often made of omitting the digitalis after the patient is relieved of heart failure; in about a fortnight the condition is again as bad as ever. Established fibrillation means digitalis for life, which should not be looked on as a punishment, but rather as a veritable elixir of life.

Warn patients that vomiting and headache may be expected and should be reported promptly. Otherwise mistaken ideas arise that they have been overdosed and poisoned, and they may refuse to continue treatment. A preparation in large doses which will not produce vomiting, is inert and should be discarded. As soon as nausea and vomiting appear, suspend treatment for twenty-four to forty-eight hours and proceed with lessened dosage. Be sure that the vomiting is not the result of heart failure itself, which will need digitalis for relief. Digitalis vomiting and headache may be relieved by a hypodermic injection of 0.65 milligramme (one one-hundredth of a grain) of atropine. In urgent cases of heart failure, when the ventricle is beating very irregularly at high speeds and dyspnoea is pronounced, then the method of the massive dose should be used. This aims at rapid digitalization of the heart in a few hours.

An adult may receive 12 cubic centimetres (three drachms) of tincture in twenty-four hours. Naturally heroic dosage needs watching, and the heart rate, not the pulse rate, must be carefully checked.

Once full digitalization is achieved small maintenance doses will be sufficient. For extremely urgent cases, in which it is imperative to slow the fibrillating heart in a few hours, the treatment of choice is either an intravenous injection of digoxin (0.5 milligramme) or an intravenous injection of strophanthin (0.56 milligramme or one one-hundred-and-twenty-fifth of a grain). Either may be repeated in from four to six hours. Do not, however, inject strophanthin if much digitalis has been recently administered. The combined effect may seriously poison the heart muscle and may be fatal.

If coupled beats appear with administration of digitalis, be warned and cease the medicine till they disappear, and then proceed with smaller doses.

Many patients can be instructed in the intelligent use of digitalis, just as they may with insulin. Regulation of the dosage is easy once the heart is under control.

Preparations of strophanthus are not reliable for oral administration. In the rare case of intolerance for digitalis use digoxin; it is less toxic and is absorbed rapidly.

**Quinidine Treatment.**—If a patient is doing badly with digitalis, it is wrong to assume that quinidine

is interchangeable with digitalis. Quinidine acts mainly on the auricular muscle, and the objective aims at ending the fibrillation and thus restoring normal rhythm.

It does this by prolonging the refractory period of the auricle and thus the circulating wave is brought up against refractory fibres and fails to be transmitted. The circus movement ceases and the normal pacemaker, the sino-auricular node, again assumes control of rhythm.

But it can be relied on to do so only in some 50% to 60% of cases, because it has other actions akin to atropine. It may lower the conduction time in the auricle; this slows the fibrillary rate, but it allows more time for the refractory period to lift and thus permits reentry of the circulating wave and perpetuates the fibrillation. It is a question which factor predominates, the effect on the refractory period or the effect on the auricular conduction.

The best cases for quinidine treatment are: (i) paroxysmal cases, (ii) recently established fibrillation, (iii) those without gross signs of organic disease and with good cardiac reserve, (iv) after thyroidectomy operations for toxic goitre.

Contraindications are found conversely in those patients: (i) with old-standing fibrillation; (ii) with gross organic disease, as in old-standing mitral stenosis; (iii) with heart failure; (iv) with recent cardiac hæmoptysis; (v) with embolic lesions; (vi) with active infection.

Remember also that even if normal rhythm is restored, the underlying pathological change still exists, so that fibrillation may recur. This is only a relative bar, because the course may be repeated and also persistent administration may avert recurrence. The chief danger from the use of quinidine comes, paradoxically, from its success. When normal auricular contractions result, thrombi may be detached and swept into the circulation, causing embolism. The risk is not great, but unpredictable, and acts as a decided deterrent to the use of this drug.

Tolerance for quinidine varies: nausea, vomiting and diarrhoea, headache, faintness and precordial discomfort may occur. If its use is decided on, a test dose of 0.18 gramme (three grains) is given. If there are no untoward symptoms or signs, my practice is to give the same dose each four hours for forty-eight hours. If unsuccessful, I give the dose every two hours for six doses. If there is no result, raise the dose to 0.36 gramme (six grains) at intervals of four hours and, if necessary, for intervals of two hours for six doses. Occasionally I have used 0.48 gramme (eight grains) per dose. The heart rate and rhythm are watched carefully. If normal rhythm be restored, the doses are spaced out to six and eight hour intervals for several days and then discontinued. Needless to say, the patient should be in bed and should remain there for a week after normal rhythm returns.

**Diuretics.**—When oedema is associated with auricular fibrillation, it may be relieved by rest and

digitalis alone. If not, diuretics are necessary, such as theobromine sodium salicylate ("Diuretin") in doses of 0.6 to 1.2 grammes (ten to twenty grains) three times a day, or theocin sodium acetate, 0.3 to 0.6 gramme (five to ten grains), three times a day. Both are inclined to produce nausea, especially theocin, although it is the more active of the two.

Nowadays, however, one relies on the synthetic mercury acetate compounds, which are excellent and non-toxic as a rule. They are represented by "Salyrgan", "Neptal" and other proprietary names. The action is enhanced by a short preliminary course of ammonium chloride or nitrate, for example, 1.2 grammes (twenty grains) in mixture three times a day for two days. Then give intramuscularly one or two cubic centimetres of the mercury preparation and repeat at four-day intervals till the oedema has disappeared. After this keep the oedema at bay by occasional injections at the first reappearance of dropsy and do not wait until it is marked. In this way I have seen patients kept comfortable for three or four years. These compounds are relatively non-toxic and may be used even in cardio-renal failure, but obviously, when the kidneys are seriously degenerated, the result will be less good than in uncomplicated cardiac failure.

One other point in the treatment of heart failure is to make use of morphine for insomnia and paroxysmal dyspnoea if the barbiturates or other sedatives fail. Rest and sleep are essential. It is not unusual in cardio-renal failure for hypnotics to cause excitement and mental confusion rather than sedation. Sometimes a combination of morphine and a barbiturate overcomes this.

#### Heart Block.

Heart block in its complete form is uncommon, but not if the lesser grades, known as partial block, are included. These comprise prolonged conduction time and dropped beats. Next come 2:1 block and other ratios, and finally complete dissociation of auricle and ventricle, each beating independently.

The clinical signs of prolonged conduction time are two, namely: (i) if a presystolic bruit becomes mid-diastolic when fresh rheumatic invasion of the myocardium occurs, it is good evidence of partial block; (ii) sometimes it causes reduplication of the first sound.

Dropped beats are associated with absent heart sounds, which distinguish them from extrasystoles. A 2:1 block gives a slow rhythm at about 48 beats per minute. Complete block is usually at about 30 to 40 beats per minute. Confirmatory signs may be manifested: (i) soft sounds of auricular systoles may be heard in the intervals between the slow beats of the ventricles; (ii) every now and then loud reinforced sounds occur when auricular and ventricular beats coincide; (iii) auricular waves may be visible in the jugular veins at the base of the neck; (iv) the occurrence of Stokes-Adams attacks.

#### Treatment of Heart Block.

Treatment comprises: (i) an attempt to treat the underlying cause, for example, arteriosclerosis, subthyroidism, syphilis; (ii) an attempt to render the heart muscle irritable, as by the use of barium chloride, mostly a useless treatment; (iii) treatment of the syncopal attacks. Patients may be revived by prompt injections of adrenaline. In prolonged seizures it should be injected directly into the heart. If there is a tendency to frequent recurrence of syncope, the attacks may be successfully warded off by the use of ephedrine hydrochloride, 0.03 gramme (half a grain), every two, four or six hours. If there are premonitory warnings, a tablet should be taken promptly. Patients should carry the remedy with them.

In complete heart block there is usually a compensatory rise in systolic blood pressure. No attempt should be made to lower this.

#### Pulsus Alternans.

*Pulsus alternans* can frequently be recognized clinically whilst taking the blood pressure by the auditory method. It is noted that there is an alternate swing in the readings by a few millimetres. Only exceptionally is it possible to detect *pulsus alternans* by palpation of the radial artery. The condition is a serious prognostic sign and is not in itself amenable to treatment.

#### OBSERVATIONS UPON CARDIAC DYSPNOEA.<sup>1</sup>

By KEMPSON MADDOX,  
Sydney.

DYSPNOEA is proportional to the ratio  $\frac{\text{ventilation}}{\text{vital capacity}}$  rather than to either function alone. As Peabody<sup>(1)</sup> has put it quite simply, "The closer the ventilation approaches the vital capacity (or maximum possible ventilation), the severer the dyspnoea". Even a moderate diminution of the maximum possible (that is the vital capacity) predisposes to dyspnoea, and anything which increases the ventilation is likely to result in shortness of breath. Pulmonary congestion primarily decreases the vital capacity, and further, can reflexly increase ventilation (especially if acute).

Dealing with cardiac failure, Weiss and Robb<sup>(2)</sup> have shown that pulmonary congestion may occur before any alteration in velocity of either the pulmonary or peripheral blood flow is detectable. Hence the early detection of cardiac failure focuses on the only early sign, reduction of vital capacity as a result of pulmonary congestion.

#### Dyspnoea on Exertion.

Dyspnoea on exertion is a physiological phenomenon applicable equally to the trained athlete and to the lounge lizard. It is only when the dyspnoea is produced in a certain individual by an amount of effort such as can be performed by the average

<sup>1</sup> Being the substance of an address to the Medical Section of the Queensland Branch of the British Medical Association, November, 1935.

untrained individual without distress that we should turn our attention to the cardio-respiratory system. It is impossible to fix arbitrary standards. Exercise tolerance tests, estimations of vital capacity, breath-holding tests and the capacity to maintain a column of mercury at a fixed elevation are all unsatisfactory in clinical practice, however suitable they may be for medical standards of health imposed upon healthy young men seeking admission, for example, to the Air Force.

Cardiac breathlessness is to be judged not by the amount of distress which it causes, but by the degree of physical effort which brings it into being. In the interrogation of a person suspected of cardiac disease, it is essential to have exact knowledge of just how much exertion produces dyspnoea and, for the purpose of prognosis, as to whether the same exertion produces the same or more dyspnoea at the present time than formerly. The practitioner living in the same locality as the patient has an advantage in that he can refer to a patient's sensations on mounting a certain hill or flight of stairs and even compare them with his own. The more ready production of dyspnoea by an accustomed exercise may, of course, be due to other factors than to progressive cardiac failure. Thus a sudden increase in weight, a recent bout of influenza or other infection, an unknown pneumothorax, pleural effusion, anaemia, pregnancy *et cetera* may be alone or partly responsible. The assessment of the cardiac factor in pulmonary disease is always difficult, and depends on a careful evaluation of the nature and extent of the pulmonary disease, with consideration of the shape and size of the heart as revealed fluoroscopically. Few people realize how many inroads upon the vital capacity and mechanical respiratory efficiency of one's college days have been made by the sedentary conditions of work and transport of today, so that we must remember the nature of a patient's work and habits in assessing his response to a given exercise. A difference of 6% mechanical efficiency in respiration serves to distinguish the athlete from the spectator, as a normal increase in ventilation on marked exercise is from five to sixty litres. There are more hypersensitive respiratory centres in the human kind today than ever before, all possessing a lowered threshold for dyspnoea. We must accept with caution a history of breathlessness from a woman with several abdominal scars. Intermittent sighing was considered by Sir William Gairdner to be a sign of fatty degeneration of the heart. We know it to be so frequently a functional disturbance that its presence is of value in differential diagnosis.

The adaptations and provisions for exercise are some of the most remarkable in nature. It is (or was) considered that the metabolites, volatile and non-volatile, of muscular contraction reaching the respiratory centre, enhanced the stimulation provided by the lessened oxygen content of the arterial blood. It was thought that persons with cardiac disease were unable to increase the output of blood from the heart sufficiently to oxidize these metabolites and maintain a proper oxygenation of the respiratory centre. Cardiac patients should have, therefore, a greater oxygen debt at the conclusion of the same exercise performed by normal individuals. Harrison

and Pilcher<sup>(3)</sup> have shown that if normal persons and cardiac patients perform the maximum exertion of which they are capable, up to the point of complete exhaustion, then the oxygen debt is much less in the case of the patient, and is indeed relatively small. The patient is checked by his dyspnoea long before he can acquire a large oxygen debt. The security for this debt is the power of the tissues to buffer lactic acid. Investigations have been made to find whether this security is diminished in cardiac failure by the presence of an acidosis. Careful blood gas analysis and pH estimation during and after exercise have quite failed to find any, either in the venous blood from the muscles or the arterial blood to the respiratory centre. This applies to persons with normal as well as diseased hearts.

We must, therefore, look elsewhere for the reason for dyspnoea, since the clinical hypothesis does not seem to have any laboratory confirmation. Even the theory that a decrease in the blood flow through the brain would allow of local accumulation of carbon dioxide in the region of the respiratory centre remains unsupported, as a result of gas analysis of blood obtained before and just after exercise. Increase in ventilation cannot then be due to a lessened circulation through the medulla. The theory that exercise produces more dyspnoea chiefly by causing further pulmonary congestion has been disproved by observations on vital capacity immediately after exercise. Harrison, Harrison, Calhoun and Marsh<sup>(4)</sup> failed to find any alteration after mild exercise in either normal or cardiac patients.

A series of ingenious experiments by Harrison *et alii*<sup>(5)</sup> seem to throw a new light on the problem. After a rest period a normal individual's respirations, as recorded on a spirometer, were found to be regular at a certain frequency. The subject then opened and closed his hands rapidly for a minute or two. Breathing increased at the moment the exercise began, too soon to be due to any chemical change, even of a hitherto unrecognized order. The same thing occurred after arrest of the circulation in the arms by sphygmomanometer cuffs inflated to 200 millimetres of mercury. The only explanation possible for this hyperpnoea is that it constitutes a nervous reflex. Study of decorticated animal preparations showed that it was not an overflow from the cortical motor centres. Further, passive movement of the subject's arms produced similar results in the respiration. We have yet to see why this reflex should cause deeper ventilation in sufferers from cardiac failure than in normal persons. A suggestion was made that perhaps a sharp increase in venous pressure, which invariably occurs on exercise, but to a high degree in congestive failure, would reflexly stimulate a deeper ventilation, just as the Bainbridge reflex stimulates a quicker pulse rate. A rapid injection of fluid of any non-toxic character into the veins had this same result. A small rubber balloon passed down the external jugular vein to the right auricle could be distended at will, and consistently deepen the ventilation as long as the vagi remained functioning.

The final physiological analysis would appear to be made up as follows:

1. There exists a primary diminution of vital capacity from pulmonary congestion. Reflex dyspnoea at rest may result from this cause alone.

2. On muscular exertion there is an immediate reflex increase in the rate and depth of respiration arising from impulses from the working muscles and, later, from the great veins close to the heart. After exercise, dyspnoea goes earlier in the normal subject because the venous pressure factor does not persist as it does for a time in the cardiac patient.

3. The increased ventilation exceeds the threshold of dyspnoea because of the existence of an already reduced vital capacity.

Chemical change then plays little part in mild exertion, but may do so in severe exertion.

#### Orthopnoea.

Orthopnoea is an important prognostic guide. Before deciding upon its presence, a certain latitude must be allowed the fat-bellied individual, with a further decrease in vital capacity from emphysema. In questioning a patient about its presence it is better to ask: (a) "How many pillows do you require at night?" and (b) "Have you had to increase the number of pillows lately?" The answer to the question "Do you lie flat on your back at night?" usually is: "No, I always sleep on my right (or left) side."

We were formerly content with the explanation that an upright decubitus relieved the diaphragm from pressure on the abdominal side, allowed the muscles a better excursion, and so hastened the return of blood through the inferior *vena cava*, which in turn increased the left ventricular output to the respiratory centre. The diaphragm, however, is a powerful structure with, perhaps, a faster metabolism than any other striped muscle, as it never rests. It is probably quite capable to overcome with ease any horizontal pressure directed upon its inferior aspect by the abdominal contents, however fat-laden. In the recumbent posture the main weight of these viscera is really upon the post-abdominal and pelvic floors. The Haldane school of physiologists held that a greater unevenness of expansion of the pulmonary fan on lying down results in arterial anoxemia, when, as a result of impaired vital capacity, an individual is unable to increase his ventilation. But investigation has failed to reveal any constant reduction in arterial oxygen saturation in orthopnoea, nor in the carbon dioxide content or tension, nor in the hydrogen ion concentration. Sir James Mackenzie subscribed to the hypothesis of diminished cerebral circulation as the origin for this and other types of cardiac dyspnoea. A certain correspondence does exist between the height of venous pressure and the degree of orthopnoea. Flexion of the head forwards on the chest may give a measure of relief; this was doubtfully interpreted as allowing of a better gravitational return of blood from the head. Withdrawal of cerebro-spinal fluid, the pressure of which is elevated in congestive cardiac failure, gives some temporary relief, suggesting that, perhaps, a better cerebral circulation ensues. Considerable clinical evidence, however, exists which opposes such a

hypothesis. For instance, the unquestionably depressed cerebral blood flow and arterial saturation accompanying severe shock or hæmorrhage is not associated with orthopnoea. Orthopnoea tends to be associated with conditions causing overfilling of the lesser circulation, especially in hypertensive failure. Many of these patients exhibit a normal venous pressure. Orthopnoea is, in general, less marked in patients with systemic venous engorgement. The symptom may occur in pneumothorax and pleural effusion, which are not specifically associated with a diminution in cardiac outflow. The symptom cannot be produced in cardiac patients without orthopnoea by artificially raising the intracranial venous pressure by pressure on the jugular veins.

With reference to the idea that rising to the sitting posture raises the amount of venous return and thus the cardiac output, Grollman<sup>(6)</sup> has shown by his acetylene technique that, while the output of blood is practically the same in the two positions, there is a momentary increase immediately after lying down.

Many observations have been made to compare the vital capacities in the two positions, lying and recumbent. The difference in the normal person averages 8%, in the patient with congestive heart failure 20%. This change, though small, would be quite sufficient to produce orthopnoea in a patient at the threshold of dyspnoea or just below it. Considerable experimental work has shown that the integrity of the vagi nerves are essential if ventilation is to increase. In an animal preparation allowing of the voluntary introduction of more blood into the pulmonary vessels, this procedure resulted in increased ventilation abolished by bilateral vagotomy. It was found that the vagus terminals were unusually sensitive to congestion of the lungs. (Harrison, Calhoun, Cullen, Wilkins and Pilcher<sup>(7)</sup>)

Simultaneous analysis of the arterial blood and blood from the cerebral venous sinuses showed that no chemical alterations occurred which could affect the experiment. Further experiments on anæsthetized dogs showed that marked alterations in the composition of the arterial blood were necessary to produce any marked increase in respiration; for example, the arterial oxygen saturation must be reduced to 70% or less; pH must be diminished to 0.12, for the ventilation to be increased by 10%. Oxygen lack and non-volatile acid affected depth rather than rate, procedures diminishing vital capacity causing shallow rapid breathing. Nervous control of respiration is, therefore, more delicate than chemical. Vagal reflexes begin at an early stage and cause respiratory changes long before malevolent influences can lead to recognizable chemical changes in the blood. Chemical regulation is primitive and is the method followed in the lowest forms of life. Nervous control is more recent and delicate, and protects against alteration in internal body chemistry. Orthopnoea, then, results in a shift of the blood from the abdomen to the chest, causing an increase of output of the right ventricle for a few seconds. It increases pulmonary congestion, decreases vital capacity, and stimulates the vagi to produce a raised ventilation. The threshold of dyspnoea is crossed

and the patient sits up, after which reverse phenomena occur. At a later stage of failure even sitting up fails to relieve the distressing and constant tachypnoea.

#### Cardiac Asthma.

Just as one is exhorted, however, to view with suspicion an apparent "idiopathic epilepsy" occurring for the first time after the age of forty, so should we regard a story of "asthma" commencing in the same decades, even in the presence of a positive family history of allergy. All grades of severity can be recognized, but even the mildest necessarily shows some "wheezing" or "tightness", as to the presence or absence of which the patient's word can usually be accepted. Occasionally a severe and even dangerous attack is the first sign of importance to indicate organic failure. By far the greater number of attacks occur at night, but not necessarily after the onset of sleep. The worst attack I have ever seen appeared in a patient who, up to this point, had had equivocal symptoms of failure, and who was still ambulatory and at work. This man stated that he had received no warning, and that the sense of suffocation and *angor animi* were indescribably horrible. Cyanosis is less common than pallor or an unpleasant earthy tinge.

Writing in his graphic way in 1897, Osler said:

The patient may spring from the bed and throw open the window in his terrible air hunger, and he assumes an attitude most favourable to the working of all the accessory muscles of respiration.

Sufferers hate to be moved, prefer to sit on the edge of the bed, and speechlessly wave their helpers away. As in all forms of asthma, a functional emphysema is present; the chest is held in inspiration, the diaphragm low, and expiratory difficulty is greatest. The pulse is small, usually regular. The blood pressure is raised, usually to a very high systolic level, an important clinical differentiation from allergic asthma. Embryocardia, gallop and accentuation of both components of the second sound are common auscultatory findings. The severer attacks produce râles at the lung bases, and may terminate in acute pulmonary oedema. There is no question but that cardiac asthma and acute oedema of the lungs (when not due to inhaled irritants) are simply stages of the same abnormal physiology, and it is this sequela which makes the administration of specific therapy such an urgency.

All clinical reviewers have noted the association of conditions imposing strain or disease predominantly upon the left ventricle with the appearance of cardiac asthma, for example, hypertension, aneurysm, aortic stenosis, coronary artery disease (which usually lays a heavier hand upon the left-sided vessel). Half the patients are hypertensives. About 20% suffer from coronary sclerosis without pronounced elevation of the arterial pressure, 20% from syphilitic heart disease (chiefly aortic aneurysm) and 6% from rheumatic heart disease. Cardiographically, no less than 17% of a series reported by Weiss and Robb<sup>(2)</sup> revealed bundle branch block of the common (formerly named) left variety. All these conditions lead to

overwork and dilatation of the left side of the heart, and are ultimately associated with the remaining components of the syndrome of left ventricular failure, namely gallop rhythm, *pulsus alternans* and cardiac pain. The general clinical picture is that of "ischemic" as opposed to "congestive" cardiac failure. Indeed, as we shall see, the development of hepatic and portal engorgement relieves the overburdened lesser circuit, with disappearance of the asthma.

For therapeutic reasons, a close study has been made of the precipitating factors of an attack. A heavy evening meal, coughing, nightmares, the desire to urinate, sudden awakening by loud noise, assumption of the horizontal position, abdominal distension, consciously or unconsciously, have been noted in many sufferers. It may be contended that coughing is a sign of the already established attack, but patients have been described who could produce an attack at will by deliberate coughing. The rise in pressure in the pulmonary circulation during coughing is presumed from the systemic venous distention which accompanies the act. Such a rise may have important reflex effects tending to initiate an attack. Some patients cough continuously and desperately throughout their seizures. Sleep-starts or a sudden awakening from an unpleasant dream finds the nervous system in a highly irritable condition by which vagal reflexes to and from the lungs are facilitated. Experiments show that ventilation is also greatly increased at such times. It must be remembered that during sleep the stimuli are allowed to become maximal before waking the patient, whereupon the nervous system changes suddenly from a variably depressed to a greatly increased state of irritability.

Few observers have discussed the elements of bronchospasm or vasodilator changes, which play such an acknowledged rôle in the genesis of allergic bronchial asthma, but the good effects of large doses of atropine (0.43 milligramme or  $\frac{1}{150}$  grain), and even of the effective but less advisable epinephrine, must act through this mechanism. Weiss and Robb<sup>(2)</sup> hold that these nervous "triggers" precipitate a sudden imbalance between right and left ventricles; Harrison *et alii*, that such a sudden rise in pressure in the already overburdened pulmonary circuit reflexly promotes bronchospasm. The Continental conception is built around the parasympathetic-sympathetic hypothesis originated by Eppinger,<sup>(3)</sup> which relates nocturnal asthma to an increase in parasympathetic tone and so to the colics and onset of labour which appear more frequently during the hours of rest.

It is interesting to speculate as to why attacks are more common during the night than following the more strenuous physical exertion of the daily work. Observations upon blood pressure during sleep, however, have revealed that it is subject to irregular and sudden rises, and by no means conducts itself at the continuously lower level followed by the pulse rate, respiration and general metabolism. While all writers are agreed upon the high, often very elevated, blood pressure found during attacks, insufficient attention has been paid, in my opinion,

to the point as to whether this is raised before, or merely follows from, the exertion and apprehension occasioned by the crisis.

A male patient of fifty-seven years was seized soon after his breakfast by severe cramping pain in the sternal region, lasting two hours and only slowly relieved by morphine. Cardiographic examination and observations on leucocytes and temperature failed to substantiate the clinical diagnosis of coronary occlusion. He had had no previous symptoms of angina. He was allowed up after three weeks in bed. No definite depreciation in his myocardial reserve was apparent. Soon afterwards he began to suffer from severe cramping pains in various parts of one leg, including the buttock, coming on after exercise. After a week the pains shifted entirely from one leg to the other. His systolic blood pressure at this time was 200, and his diastolic pressure 100 millimetres of mercury. Examination of the affected limb after deliberate exercise in my consulting room showed a sudden development of ischaemia of the foot. A week later he had a severe attack of cardiac asthma, in which his systolic blood pressure was 260 and his diastolic pressure 110 millimetres of mercury. He responded rapidly to morphia and oxygen, and his blood pressure readings next day were 160 and 100 millimetres of mercury. A mild degree of arterial thickening of his radial and temporal arteries was undeniable.

My interpretation was that the patient was a sufferer from so-called "spastic hypertension" and that probably a vasospastic crisis precipitated a left ventricular dilatation, and so cardiac asthma.

The Vienna school advances some shrewd arguments against a left-sided cardiac weakness being the whole and sole explanation for cardiac asthma. If mere overfilling of the pulmonary circuit is the main reason, why, they ask, does it not occur more often in "compensated" mitral stenosis, where pulmonary congestion attains its highest degree? Again, if primary failure of the left ventricle is responsible, why does the systolic blood pressure not fall rather than rise? Indeed, so rarely is it much diminished that one is justified in expecting a fatal issue in such circumstances from coronary infarction or grave myocardial asthenia. Morphine is the best drug available. Morphine has no specific sustaining effect upon the left ventricle. The drug acts like a charm even in the smallest doses. Surely this can only come from a depression of general nervous excitability, especially of the medullary centres. We know, further, that conditions disturbing intracranial pressure can sometimes incite an attack of paroxysmal dyspnoea, even terminating in pulmonary oedema. Acceptance of the hypothesis that the central nervous system and its pulmonary connexions are the chief actors in the drama explains also the independence of attacks upon the grade of pulmonary congestion, and that the venous pressure and peripheral blood flow remain fairly normal.

The question as to whether the absorption of fluid from the tissue spaces with presumable increase in blood volume plays any part in distending the pulmonary vessels has been raised as a result of the beneficial action of diuretics of the purin series. No change has been noted in haemoglobin concentration which would aid such a theory. The value of such diuretics is probably rather to be related to their probable capacity to increase coronary flow. Once the attack has started, observations show a loss in water content in arterial as compared with venous blood, indicating a loss of fluid into the lungs.

Careful searches by Harrison have failed to reveal any constant deviation from normal in the oxygen saturation of samples of blood from the carotid artery and jugular vein. Dyspnoeic patients have values for oxygen utilization which are within the range of those found in normal persons. Hence diminished flow of blood through the brain is not responsible. This is as close as we can go at present to the actual gaseous exchanges in the respiratory centre. Many animal experiments have produced pulmonary oedema, but are mostly unconvincing. We know little of tissue alterations from observations upon mixed venous blood. As we have already seen, increased hydrogen ion concentration of the arterial blood is an uncommon finding in cardiac dyspnoea, and to this cardiac asthma is no exception. Indeed, a mild alkalosis is a much more commoner finding.

A study of the chest and respiration between attacks reveals a similar series of findings to that discoverable between bouts of bronchial asthma. The vital capacity is much diminished and resting ventilation increased. Vascular congestion with increased hilar shadows is demonstrable fluoroscopically. The diaphragm is low, and its excursion diminished. The pulmonary second sound is accentuated or even accompanied by a systolic murmur. The movement of the right side of the heart exceeds that of the left side in the left oblique position.

Weiss and Robb were able to show that the mean velocity of pulmonary blood flow is usually decreased. Injection of dye into a peripheral vein and collection from a corresponding peripheral artery showed a variably prolonged appearance, indicating marked differences in the velocity of blood flow through the various pathways of the pulmonary circuit.

We see, then, that cardiac asthma is a complex problem. At one extreme are patients with advanced failure of the left ventricle with distended and rigid lungs (*Lungenschwellung und Lungenstarre*) in which the lung blood depôts are full to the utmost and where the balance is readily tipped in the direction of another attack, by a relatively innocent "feather", which may act from the cerebrum through the vagus or result from a small but sudden addition to the pulmonary lake, for example from slipping into the horizontal position during sleep. At the other extreme are patients with a minor grade of failure but whose irritable nervous system allows of sudden coronary spasm, together with a spontaneous extra load on the left ventricle from general vasoconstriction. The quick assumption of the sitting posture and the passing of the initiatory stimulus are favourable to the termination of the attack, but any over-ventilation increases the venous return to the heart and lungs. If the latter predominates, the vicious cycle so instituted ends in oedema of the lungs as soon as the rising intracapillary pressure exceeds the osmotic power of the proteins. Morphine acts by counteracting over-ventilation from depression of the respiratory centre; venesection or binding the limbs, by delaying venous return.

Cardiac asthma is always serious—forty sufferers out of eighty-seven were dead within twelve months.

### Cheyne-Stokes Respiration.

Relative failure of the left ventricle is preeminent in the aetiology of the extraordinary respiratory phenomenon known as periodic breathing. Even when occurring in uræmia, it is probably a part of the cardio-vascular symptomatology rather than that of the altered blood chemistry. Though a common phenomenon, it is more often unobserved. "Seek and ye shall find" Cheyne-Stokes breathing surprisingly often among your patients with hypertensive failure. One reason why it may be overlooked is the fact that it occurs in its most perfected form while the patient is dosing, and disappears during wakefulness or deep sleep. The doctor's morning visit, then, is not the appropriate hour to observe this important sign. The observer must be prepared to record its presence in a much less fully organized form than that which Stokes<sup>(9)</sup> described in these words:

For more than two months before death this singular character of respiration was always present, and so long would the periods of suspension be that his attendants were frequently in doubt whether he was not actually dead. Then a very feeble, barely perceptible inspiration would take place, followed by another somewhat stronger, until at length high, heavy and even violent respiration was established, which would then subside till the next period of suspension. This was frequently a quarter of a minute in duration. . . . I had little doubt that this was a case of weakened and probably fatty heart, with disease of the aorta.

Often there are no periods of apnoea, but merely a successive deepening and lightening of the ventilation. If such be noticed on the daily visit, one can assume safely that it will become much more accentuated at night. The nurse may merely report the patient as sleepless, stating that he will often attempt to get up or move restlessly about the room. The report may be that he wakes or stirs every few minutes. This results from the hyperpnœa, the patient dozing again between whiles. All that the patient realizes is that he is sleepless and restless for a reason he cannot understand. It is important for the medical attendant to recognize this form of sleeplessness, which is to be countered by digitalis rather than barbiturates.

Until recently the cause of periodic breathing has been considered to be unquestionably bound up with alterations in the blood gases. During the long periods of apnoea, the alveolar carbon dioxide tension progressively mounted to a high level, which, at a certain threshold, produced hyperpnœa of a grade which washed out too much of the carbon dioxide and so induced a further cessation of breathing. Oxygen lack can cause periodic breathing in the normal person. Recent investigations, however, have failed to find any constant change in arterial blood gases, either oxygen or carbon dioxide, at any phase of the completed respiratory cycle. The results of estimations of pH have been normal or on the alkaline side. Many patients with periodic breathing have arterial blood which is under-ventilated as regards oxygen and over-ventilated as regards carbon dioxide, quite the reverse to what has always been imagined. The average arterial carbon dioxide is reduced, that is the hyperpnœic predominates over the apnœic phase, which is what one would expect clinically from a periodic deepening of respiration

without apnoea. Administration of carbon dioxide will abolish Cheyne-Stokes respiration, while over-ventilation with air prolongs it; thus any disease process which tends to cause over-ventilation and consequent loss of carbon dioxide will tend to produce periodic breathing. Oxygen lack at high altitudes probably causes Cheyne-Stokes respiration by a simultaneous diminution of carbon dioxide tension in the pulmonary alveoli.

In cardiac patients anoxæmia cannot be the chief cause, because it is not invariably present, and inhalation of oxygen does not constantly abolish the phenomenon. In pulmonary disease extreme cyanosis may be present, but with an oxygen saturation as low as 50% to 60%, without irregularity of breathing. The blood of these patients cannot be aerated properly, and so carbon dioxide accumulates. Upon the only occasion on which I tried to abolish Cheyne-Stokes respiration by the administration of oxygen, the breathing was modified and quieter and the cyanosis lessened, but the periodicity remained. Dogs caused to overbreathe into a spirometer filled with oxygen failed in every instance to breathe periodically afterwards, whereas when air was breathed periodic breathing was produced in almost every animal. In so far as the initial apnoea was longer with oxygen than with air, it is an indication that the next stimulus to respiration comes from lack of oxygen rather than reaccumulation of carbon dioxide.

King and Harrison<sup>(10)</sup> have shown that arterial oxygen saturation diminishes slowly, then quickly, during the period of apnoea and the beginning of hyperpnœa, after which it is rapidly restored to normal. Carbon dioxide accumulates rapidly, and then slowly, and falls away immediately breathing commences. These differences are dependent upon varying rates of dissociation peculiar to the two gases.

Studies of the pH of the arterial blood have shown that at the beginning of apnoea the blood becomes more acid, towards the end of apnoea there is a slight return towards alkalinity, probably dependent upon conversion of oxyhæmoglobin to the more alkaline hæmoglobin. At onset of breathing a rapid fall in carbon dioxide content produces a further alkaline reaction, but a certain acid peak occurs almost at once from the sudden reoxygenation of the hæmoglobin. The reason for the crescendo nature of the hyperpnœa is this increase in acidity reaching its peak several seconds after the onset of breathing. The effect of these irregular changes in carbon dioxide tension, and in pH produces overshooting of the respiration in one or other direction and thus provides for the continuance of the attack.

Eyster<sup>(11)</sup> discovered that the changes in respiration were preceded by periodic alterations in the blood pressure which passed above and then below the level of intracranial pressure. He cited this as a possible cause of rhythmicity, but subsequent work has shown that blood pressure changes are of a nature of results rather than causes. Harrison<sup>(10)</sup> suggests that changes in the pH of the respiratory centre are a result of its own activity and that irregular neutralization may play a part.

To sum up, primary overbreathing and the onset of sleep seem to be sufficient to initiate Cheyne-Stokes respiration in cardiac disease. The over-ventilation performs this by diminution in the carbon dioxide tension of the arterial blood, resulting in apnoea. The effect of drowsiness must be to prolong the apnoea sufficiently to allow the chemical changes described above to operate. The inevitable movements of some muscles or other when the patient is awake would produce reflex stimulation of breathing and cut the apnoeic period too short to allow of any overshooting. When sleep becomes deeper, however, the depressed respiratory centre no longer produces a high degree of hyperpnoea. The average carbon dioxide concentration gradually rises and clinically obvious periodicity goes. Cheyne-Stokes respiration is, therefore, dependent on lack of coordination between features which normally control breathing. Pulmonary congestion produces reflex stimulation of breathing. Carbon dioxide is lost and a paradoxical condition follows in which the blood is too much aerated as regards carbon dioxide and too little aerated as regards oxygen. A waxing and waning respiratory distress follows.

#### Dyspnoea due to Complications.

The sudden diminution of vital capacity due to hydrothorax or pneumothorax lowers the dyspnoeic threshold and produces reflex respiratory stimulation. Ascites and meteorism are causes of similar pressure on the lungs and reduction of the area available for aeration, this time by compression from below. Pericardial effusion can similarly reduce lung volume, and probably acts in this way more than by an interference with diastolic filling of the heart. Pulmonary infarction causes reflex dyspnoea and, again, a lessening in the size of the respiratory membrane.

What part does the raised basal metabolic rate of cardiac failure play in the production of dyspnoea? The raised basal metabolic rate, which may reach as much as 25%, is usually said to be the result of the muscular effort of increased ventilation. Experiments showed that artificial stimulation of ventilation by the breathing of carbon dioxide is associated with a marked increase in oxygen consumption, that is in basal metabolic rate. This increase was abolished after the use of morphine, so that this drug acts by decreasing both oxygen consumption and cardiac output. It is impossible to determine satisfactorily whether the increased oxygen consumption of the heart itself contributes at all to the metabolic rise. Dyspnoea is, therefore, often wasteful and unnecessary; it raises the cardiac output, increases the pulmonary congestion, provokes the greater requirements of oxygen; when continuous, it interferes with sleep and the patient wastes into the state of cardiac cachexia. It may lead to morphine addiction. The individual at rest cannot protect himself by resting more completely. Dyspnoea for him is by no means beneficial or compensatory, but merely aggravates his misery.

#### Symptomatic Therapy.

Rest combined with digitalis in effective dosage and in an active absorbable form is, of course, the

fundamental treatment for cardiac dyspnoea. By means of digoxin it is possible to make its benefits apparent in fifteen to twenty minutes. Oxygen, best given by nasal catheter or Davies-Gilchrist mask, is very helpful, especially in continuous dyspnoea and cardiac asthma. A properly constructed oxygen tent or chamber with provision for cooling circulation, carbon dioxide absorption, and drying of the contained atmosphere is, as yet, unobtainable in this country. Oxygen, we have seen, provides some relief for Cheyne-Stokes attacks, also. Much better, however, is euphyllin (aminophyllin, corphyllamine), a combination of theocin and ethylene diamine (as solvent). Administered intravenously, it causes a general vasodilatation, but its good effects are due to more than this result. In a dose of 0.25 gramme diluted with fifteen to twenty cubic centimetres of 30% glucose, or given alone or very slowly, so that at least three minutes are taken over the injection, the drug can be repeated indefinitely. Idiosyncrasies are rare. Subcutaneously or intramuscularly, it causes local pain. It is absorbed *per rectum* (dose 0.48 gramme in fifty cubic centimetres of saline solution), but it is useless when given by mouth. Combined with "Medinal", chloral or a small dose of morphine, "Omnopon" or heroin, it is a good prophylactic against a night disturbed by periodic dyspnoea. Large doses of morphine are contraindicated; though they lessen the violence, they prolong the duration of the attacks, which themselves sometimes are a feature of morphine poisoning.

Morphine, however, is the drug of selection for cardiac asthma. It should be ordered boldly (0.02 gramme, or a third of a grain), and given intramuscularly or even intravenously if the capillary circulation is bad. Atropine sulphate is also of value in full doses, and may be used as a prophylactic; or a small dose of heroin, on retiring. Venesection is called for in prolonged or very frequent attacks, a full pint of blood being taken. The procedure relieves the high systemic blood pressure, as well as the pulmonary congestion. Bloodless venesection by bandaging the proximal parts of the limbs is said to be of benefit, and can be used for half an hour in the evening as a preventive. I have no experience of the use of pituitrin, adrenaline or injections of 40% to 50% grape sugar.

Orthopnoea is best treated in a Lewis bed or, failing this, by raising the foot of the bed twelve inches and arranging a bolster under the knees.

#### Conclusions.

In an age of medicine for ever striving for earlier diagnosis the cardiologist is at a disadvantage. Dyspnoea, the earliest symptom of heart disease, in its milder forms, is tolerated by the patient, who contents himself with the philosophy of increasing years. When the physician is consulted, the cardiac reserve is already low. His is the difficult task of assessing what remains, of steering a course between pessimism and over-optimism, detecting or avoiding the addition of the fearful cloak of neurotic anxiety. We see that we must now regard the very symptom which is the first and chief fingerpoint to cardiac breakdown, as more intimately bound to the nervous

system and all its vagaries than to the complex glassware of the laboratory bench.

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### THE ROLE OF THE ROYAL AUSTRALASIAN COLLEGE OF SURGEONS IN POST-GRADUATE EDUCATION.

By ALAN NEWTON,

Censor-in-Chief, Royal Australasian College of Surgeons.

WE have just been privileged to listen to addresses from two men whose judgement we cannot afford to ignore. The President of the Royal College of Surgeons of England, who has a wide knowledge of, and great experience in, the field of post-graduate education, has placed us even more deeply in his debt by his address, which will be of the greatest value to the Council of the Royal Australasian College of Surgeons in the discharge of its important duty of organizing post-graduate surgical education in Australia and New Zealand. Professor Saint, who, as you know, was a distinguished member of the famous Newcastle school of medicine before he was appointed to the chair of surgery at Cape Town, and was therefore a disciple of Rutherford Morrison, one of the greatest surgical teachers of modern times, has dealt with certain problems of undergraduate education in a manner which has earned the gratitude of his audience, more particularly those of us who are members of the faculties of medicine of the Australasian universities.

It is appropriate that addresses on both undergraduate and post-graduate work should have been given tonight, because these two periods of training must be closely connected. Any distinction between them is artificial and is due to the fact that universities must be concerned primarily with undergraduate education and have not functioned to a great extent, except as examining bodies, in

the domain of post-graduate surgical training. We feel proud of our medical schools and believe that the standard of undergraduate training in Australia and New Zealand has reached a high level; but it is generally recognized that, owing to the great increase in the mass of medical knowledge, undergraduate training must be mainly confined to the study of general principles and must be followed, particularly in the case of those men who desire to specialize, by a period of post-graduate training. The need for the organization and control of post-graduate training in surgery was one of the chief reasons for the foundation of the Royal Australasian College of Surgeons eight years ago. It was realized that this could be better accomplished, throughout Australia and New Zealand, by the formation of an Australasian college, directed by surgeons, than by requesting each university, already overburdened by teaching work, independently to develop and to extend its activities in this direction. It is obvious that there must be the closest cooperation between the College, the universities and the large hospitals, as the activities of these bodies necessarily overlap to some extent. It is therefore a pleasant duty to acknowledge with gratitude the great help that our young College has been given in its educational work by the universities and by most of the large hospitals in Australia and New Zealand.

The rôle of the College in post-graduate education must embrace the education of its Fellows, the education and examination of candidates for its Fellowship and the provision of educational opportunities to other members of the medical profession.

The College is not content merely to bestow a diploma upon those deemed worthy of its Fellowship. The education of a surgeon must continue throughout his lifetime. The College therefore arranges an annual general meeting in one of the various Australasian cities, at which some days are devoted to operative and clinical demonstrations and lectures. It also arranges numerous other meetings in the various States and in New Zealand. It is a point of honour for its Fellows to attend as many as possible of these meetings. In this way surgeons continually learn, not only from their colleagues, but also, as on this occasion, from distinguished visitors. There is no doubt that as a result of this policy the general level of surgical work in Australasia has been appreciably raised since the foundation of the College. The publication by the College of *The Australian and New Zealand Journal of Surgery* has also been of great value in this direction.

Candidates for Fellowship of the College must undergo a course of training extending over a minimum period of five years after graduation, followed by an examination by a board of censors. It is unnecessary for me to describe the requirements in detail, though some unique features deserve emphasis. All candidates must serve a period of surgical apprenticeship additional to the time spent as a house surgeon, in the wards and operating theatres of an approved hospital, under the super-

vision of a senior surgeon, for it is believed that this training is essential for the development of surgical judgement and technique. Apart from this requirement, the regulations are not hidebound, for it is possible for each candidate to vary his training in accordance with his desire and opportunities, due credit being given for work in other countries, in research and in the various departments of university medical schools. No candidate can present himself for examination until his training has been approved. The College recognizes that the possession of a senior surgical qualification as granted by most universities and colleges, does not invariably mean that the recipient is a capable operating surgeon, but, as such a qualification indicates at least a sound training in the basic principles of surgery, it forms part of the requirements for Fellowship of the Royal Australasian College of Surgeons. The period of surgical apprenticeship in surgery should follow the acquisition of a degree such as the Master of Surgery, for which, in the Australian universities, the candidate can present himself in two or three years after graduation. With your permission I shall digress for a moment to point out that, when the College was founded, the regulations governing admission to these degrees differed widely in the three Australian universities. Thanks to the cooperation to which I have already referred, uniform regulations were introduced in these universities some years ago, which provide that all candidates shall pass a primary examination in anatomy and physiology and a final examination in surgery. The College therefore defined a senior surgical qualification in its regulations as one which embraced these two examinations. The interests of universities and colleges in which only one examination was demanded for a senior surgical qualification were safeguarded by the proviso that the two examinations need not necessarily be passed in the same university or college.

The provision of opportunities for men to undergo an adequate training is of much greater importance than drafting regulations and conducting examinations, so that I shall now describe what the College has done in this direction. It was necessary to create positions, of the surgical apprentice type, for men who had completed their periods as resident medical officers at hospital and had obtained a senior surgical qualification. When approached by the College, the authorities of nearly all the large clinical schools in Australia, willingly cooperated by making available positions of this nature, tenable for periods of two or three years. In the last two years twelve men in Melbourne alone have undergone this training.

A step of even greater importance in this direction is the agreement between the College and the Committee of Management of Prince Henry's Hospital, Melbourne, whereby this institution will be rebuilt and will become a great surgical post-graduate clinical school. You are aware of the fact that, throughout the world, the greatest significance is

attached to the establishment of post-graduate, in addition to undergraduate clinical schools, and it is gratifying to feel that we are not deficient in this requirement. It is of paramount importance to the community that its surgeons should be adequately trained and a large hospital should be specially devoted to this purpose. The staff of the hospital will be assisted in teaching by lecturers nominated by the College, which proposes to ask surgeons from other States and other countries to share in this work. It is to be hoped that Fellows generally will do everything possible to assist the appeal for funds for rebuilding the hospital, in order to show their appreciation of the wisdom and forethought of its committee of management.

Six years ago the Royal Australasian College of Surgeons approached the Royal College of Surgeons of England with a request that the primary examination for fellowship of the latter college should be held in this country. As you know, our request was granted, and an examination was held in Melbourne in 1931 and again in Melbourne and Dunedin in 1934. I hope that you, Sir, as President of the Royal College of Surgeons of England, will convey to your Council our gratitude for its action and also our hope that it will be possible to repeat the examination in this country at regular intervals. Holding the examination here is not only of imperial significance, but also has the advantage that successful candidates must go to London to sit for the final examination and thus be brought under the influence of the great traditions of British surgery. The examination is a potent stimulus to both undergraduate and post-graduate work. Viewed from another angle, an examination conducted by an outside body constitutes a test of the work done in a teaching institution, and I think that our universities should be congratulated upon the fact that 50% of candidates were approved on each occasion, a much higher percentage of successes than is usual in this examination.

As a further inducement to travel the Council has established a bureau of information concerning the opportunities available for post-graduate work in other countries.

The Council wishes to encourage post-graduate students to work in university departments and believes that, as the number of candidates for its Fellowship increases, the supply of workers of this type will also increase.

I must be brief and must now refer to another aspect of the problem of post-graduate education. It is obvious that the addition of a five-year period of post-graduate training to a six-year period of undergraduate training must deter many men, on economic grounds, from qualifying as well trained surgeons. The College proposes to devote a large part of the income from the Gordon Craig bequest, of approximately £60,000, when it becomes available, to assist able men to undergo this training. Further, now that its building is completed free of debt, it proposes to devote portion of its income to this purpose, for it is appropriate that the dues

paid to the College by surgeons of today should be devoted, in part, to the education of the surgeons of tomorrow. The Syme Research Scholarship, being portion of the income of £2,500 given to the College by his family in memory of Sir George Syme, our first President, and two Ryan Scholarships, each of £100 annually, given by a Fellow, T. F. Ryan, in memory of Michael Ryan and J. P. Ryan, available to students graduating from the Saint Vincent's and the Melbourne Hospital clinical schools respectively, will also help materially in this direction. It is significant and gratifying that the College in its short life has already received so much help in its work.

The College must also study the needs of others than its Fellows and candidates for its Fellowship. There are many men who graduated prior to the foundation of the College and who wish to undergo further surgical training of a type not available to them immediately after they had graduated. Opportunities will be afforded to such men to undergo this training at Prince Henry's Hospital, even though it may not be possible for them to take the full course of training for Fellowship. This policy will "bridge over" the transition period necessary before the advent of a time when we hope it will be customary for all those who practise surgery to accomplish the long course of training demanded of candidates for Fellowship of the Royal Australasian College of Surgeons.

It is a cherished tradition of our profession that we should continually strive to improve the work we do. We are proud of the general level of medical and surgical work in Australia and New Zealand, but we must realize that:

To have done is to hang,  
Quite out of fashion, like a rusty mail  
In monumental mockery.

We must go forward. Professor Wood Jones, in his eloquent Syme Oration, suggested that we may be now in a transition phase in surgery, and that we might in future take one of two roads in surgical development. Let us hope that the College, seeking and receiving the support and encouragement of the universities and the general body of the medical profession, may take the right road in its rôle of directing surgical post-graduate education in this country.

## Reviews.

### GRAY'S ANATOMY.

THE twenty-sixth edition of "Gray's Anatomy" is a reminder that the science of human anatomy is by no means static.<sup>1</sup> Just over two years ago we reviewed the

twenty-fifth edition and remarked on the excellent presentation of the subject. The appearance of another and yet more elaborate edition at this early date speaks volumes for the industry and the enthusiasm of the editor, Professor T. B. Johnston, and is a fitting monument to Henry Gray.

The original author was the son of a private messenger to King George IV. He learned his anatomy by the slow and painstaking method of making dissections himself. While still a student he secured the triennial prize of the Royal College of Surgeons for an essay entitled "The Origin, Connections and Distribution of the Nerves of the Human Eye and its Appendages, illustrated by Comparative Dissections of the Eye in other Vertebrate Animals". At the age of twenty-six he won the Astley Cooper Prize for a dissertation, "On the Structure and Use of the Spleen". The standard he set in the original text-book has been faithfully maintained by his successors.

Gray's original work contained 363 figures; this number has been increased to 1,323 in the present edition. The popularity of the text-book undoubtedly owes much to the coloured illustrations which have always been a feature of the book. Perhaps another reason for the publication of this new edition is the recent change in nomenclature. Readers need not be unduly alarmed.

In June, 1933, the Anatomical Society of Great Britain and Ireland approved the adoption of the British revision of the B.N.A. This revised terminology has been employed in the new work. Whatever older surgeons may think of the frequent changes in nomenclature in recent years, anatomists certainly are seeking to simplify the subject by revising the terms used in the description of anatomical structures. This effort is to be applauded, especially in these days of an overloaded medical curriculum. Some typical changes in the British revision of the B.N.A. are as follow: The scaphoid (B.N.A., navicular) bone of the wrist is distinguished from the navicular bone of the foot. Trapezium and trapezoid replace the B.N.A. greater multangular and lesser multangular respectively. The hybrid terms white *rami communicantes* of the B.N.A. are supplanted by the English white communicating branches. The entirely unrelated term *lacertus fibrosus* becomes the bicipital aponeurosis. The *ligamentum carpi transversum* is more physiologically described as the *flexor retinaculum*. The B.N.A. ileo-pectineal bursa is more correctly defined as the bursa of the psoas major tendon.

Perhaps the terms are less euphonious, but they are of more practical value. Certainly the term trochlear notch is a better aid to memory than the rather vague B.N.A. term, *incisura semilunaris* of the ulna.

Future generations of medical students will benefit by the change of nomenclature, but there will be a few regrets at the passing of some of the well worn familiar classical names. The sponsors of the old terminology may take consolation in the fact that some of the old terminology names have been restored.

The book appears so complete that comment seems fatuous. The repeated neglect of such important regions as the fascial spaces of the hand requires more explanation than that they are not obvious in the cadaver. The thyroid and parathyroid glands are relegated to relatively little space in the book. The warning that "removal of one lobe [of the thyroid] with division of the isthmus may be required in parenchymatous goitre . . . is a radical proceeding and carries a much greater risk of hæmorrhage" is possibly a relic of previous editions, but indicates the need for revision of the applied anatomy sections of the work. No mention is made of the valuable contribution of Walton to the topography of the parathyroids.

The osteological section is especially well done. The descriptions of the skull as a whole are more useful to students than the detailed study of the individual bones. The contributions of radiology in recent years, particularly in the region of the lumbo-sacral portion of the spine,

<sup>1</sup>"Anatomy, Descriptive and Applied" by H. Gray, F.R.S., F.R.C.S.; Twenty-Sixth Edition, edited by T. B. Johnston, M.B., Ch.B.; 1935. London: Longmans, Green and Company. Royal 8vo, pp. 1548, with 1,323 illustrations, of which 611 are coloured. Price: 42s. net.

have received scant attention. It is difficult to decide what is normal in this part of the spine, judging by the frequent finding of abnormalities in the articulations of the lumbar and sacral spine. As an attempt is made to present applied anatomy in the text, the inclusion of a little of this new matter would be welcome.

The development of the genitalia includes much new work and many difficulties are cleared up. This section of the book has always presented an excellent account of embryology and is quite up to standard in the incorporation of the accepted results of recent research. The elementary histology, so beautifully illustrated, is a feature which has always enhanced the value of "Gray's Anatomy". There is a unity about the book.

In one single volume the macroscopic and microscopic structure of the normal human body are described in sufficient detail, with a few exceptions, to justify the title of this last edition as "the student's friend and the practitioner's companion".

Professor Johnston is to be congratulated on his achievement. For no other anatomical text-book is the term *vade mecum* so apposite.

### CIRCULATORY FAILURE.

THE name of Harrison and his associates has appeared so frequently in original contributions of merit describing valuable additions to experimental cardiology that a complete and concise summary of his work to date has been awaited with interest. "Failure of the Circulation" fulfils our best anticipations, as the book presents in a concise, easily readable and well arranged fashion, a fair and unbiased review of modern physiological and pharmacological investigations concerned with the cardio-vascular system.<sup>1</sup> Each piece of research is illustrated by well drawn figures or numerical tables transposed from the original article itself. Summaries of previous work and finely technical details are printed in small type so that they may be omitted by a reader interested only in the main or clinical theme. A logical presentation of the argument, short chapters, each rounded off by a summary and a final chapter epitomizing all the others, with a full bibliography of nearly three hundred references, reveal an understanding of the factors which make for good medical authorship.

The burden of Harrison's argument is a suggested reclassification of circulatory disturbances. While deploring the introduction of new and ugly terms into the cardiological vernacular, the reader, by the time he has completed his task, is inclined to realize their value for purposes of better diagnosis and teaching.

For disturbances of the peripheral circulation of the nature observed in shock following myocardial infarction, dehydration or injury to the central nervous system, Harrison would have us employ the term "hypokinetic". This class of disorder may be further subdivided according to its origin into types named hæmatogenic, neurogenic, vasogenic and cardiogenic. The clinical syndromes, commonly known as "irritable heart", "effort syndrome", "neurocirculatory asthma" and the circulatory phenomena of tachycardia, loud heart sounds, forcible cardiac movement and increase in pulse pressure found in hyperthyroidism or anemia are grouped together under the term "hyperkinetic syndrome". Congestive cardiac failure and ichæmic cardiac disturbance are preferably described as "dyskinetic". It is a difficult task, as many reformers have found, to alter well known terminology overnight, however desirable such a change may become. We think that the titles suggested are of distinct advantage. Many intermediate groups occur in the clinical

field, and chapters are devoted to a description of the dyskinetic phenomena in association with either hyperkinetic or hypokinetic features.

The book commences with a full clinical description of each type. The next section deals with the cardinal symptoms and signs of circulatory failure. The best feature of this part is that concerned with dyspnoea. This is to be expected, as it is this symptom which has been the subject of ten years' close investigation at the hands of the author and his colleagues. A classification of considerable clinical value is outlined, and the mechanism of each type is thoroughly probed. The "ventilation index", which is an attempt at a quantitative objective evaluation of the syndrome, and which takes account of the height and weight of the subject, is asserted to be of small value in differential diagnosis, but to have distinct function in the recording of clinical progress in any one individual.

The major part of the work deals naturally with the dyskinetic syndrome, and physiological researches are introduced to support almost every phase of clinical therapy. A logical and convincing argument, including tables of cardiac output measured by the latest Grollmann technique, establishes beyond doubt the better claims of the "backward", as opposed to the "forward failure", hypothesis of the mechanism of cardiac failure in general.

The advantages and disadvantages of cardiac enlargement to the patient so afflicted make interesting reading. The dilated heart requires more oxygen. If this cannot be supplied fatigue occurs, which is the fundamental factor in cardiac inefficiency. With the author's plea for a limitation of the term *angina pectoris* to the term of Heberden's definition all thinking physicians have long been in agreement. This diagnosis "angina" implies a liability to sudden death; this is inseparable. To play with terms such as "pseudoangina", "false angina" *et cetera* is to betray either diagnostic incompetency or pitiless unconcern for a patient's mental tranquillity.

The final quarter of the book is devoted to the prognosis and treatment of congestive heart failure. The main facts in prognosis and in the differential diagnosis of organic from functional cardiac disorder are succinctly stated. Harrison, like the majority of his compatriots, prefers whole leaf digitalis to the tincture, as it is supposed to contain the coronary dilator, digitonin. He speaks highly of the value of euphyllin in the treatment of Cheyne-Stokes dyspnoea. He utters a warning against the possibility of ventricular fibrillation during digitalization after myocardial infarction and in the presence of peripheral circulatory failure, or in acute myocarditis. The old question of the effect of digitalis upon the cardiac output has been personally reinvestigated with equivocal results. He warmly recommends small doses of the drug during the preœdema phase of cardiac defeat, or even prophylactically under certain conditions. The use of "Salyrgan" is also praised during this period. An ambulatory patient weighs himself daily; any sudden addition observed on the scales is countered by one or two intravenous injections of the drug. "Diuretin" is damned with faint praise, a conclusion with which many workers will agree. Potassium chloride has more biochemical advantages, even in congestive failure, and may be used to pacify the oedematous subject's craving for salt, or given as potassium dibasic phosphate, two grammes thrice daily. American physicians have more success than others in the management of coronary thrombosis, as they keep their patients at absolute rest for at least six weeks, on a low 800 to 1,000 calories diet with plenty of carbohydrate.

Dr. Harrison's contribution is so comprehensive that it is difficult to find any gaps or inadequately discussed aspects of this important subject. With the exclusion of rheumatic heart disease and the arrhythmias, he has almost written a text-book, but better and more practical. Vaso-vagal disturbance is probably a term to be included under the hypokinetic syndrome. The treatment of this and of neurogenic circulatory disturbances in general might perhaps have been dealt with more fully. In the first part of the book there are frequent misprints, even allowing for the vagaries of American spelling.

<sup>1</sup> "Failure of the Circulation", by T. R. Harrison, M.D.: 1935. London: Baillière, Tindall and Cox. Royal 8vo, pp. 377, with illustrations. Price: 20s. net.

## The Medical Journal of Australia

SATURDAY, JANUARY 25, 1936.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

### THE CHRONIC RHEUMATIC DISEASES.

FEW conditions in the whole range of medicine have caused a greater divergence of opinion than what are known as the chronic rheumatic diseases. One type of worker, either from force of habit or for the sake of convenience, applies the term rheumatism to almost any vague condition characterized by pain of a certain type; another, striving for accuracy, would dispense with the term and would use only terms that can be justified by a knowledge of pathology. The latter would be the more admirable attitude, were it not that, owing to gaps in our knowledge, those who adopt it are compelled either to leave some conditions unnamed or to give them names ill-founded or obviously incorrect. There is some justification for the retention of the term rheumatic, provided it is retained, not as a cloak of ignorance, but as a term of convenience, as the lawyers would say "without prejudice", until such time as pathological insight becomes greater. This view is adopted on the

Continent of Europe, where we find a *Revue du Rhumatisme*, an International Congress for the Study of Rheumatism, and the *Ligue Internationale contre le Rhumatisme*. The view has also been adopted in Great Britain.

As a result of a suggestion made at a meeting of the *Ligue Internationale contre le Rhumatisme* in 1932, the Royal College of Physicians of London appointed a National Committee on Chronic Rheumatic Diseases. Sir Humphry Rolleston is the Chairman of the Committee, and Dr. W. S. C. Copeman is the Honorary Secretary. The Committee held its first meeting on March 16, 1934; at its third meeting the Committee decided that for a trial period of three years it should publish an annual report and that this report should include a critical abstract of the year's literature. The first report has appeared.<sup>1</sup> The editor, Dr. C. W. Buckley, states in the preface that the object of the Committee in publishing the report is to bring to the notice of those interested in the subject of chronic rheumatism recent clinical and laboratory research work. He thinks that much of the work being done in England does not gain the publicity that it deserves, for want of a suitable channel through which it may be made known. In view of the many excellent journals published in England, this is a little hard to understand. However, this report will be welcomed as setting out under one cover the recent work that has been done on the subject. In drawing the attention of readers to this work, we would urge those interested in the subject to secure a copy for themselves; they will be amply repaid.

There has been no attempt in this book to reproduce a "majority report". Sir Humphry Rolleston makes this quite clear in his foreword; he thinks that truth is more likely to emerge from the unfettered expression of ideas and from their free discussion than from any attempt to force them into a common mould. An interesting sidelight is thrown on majority reports by Dr. John Freeman, who discusses allergy and hypersensitiveness. He

<sup>1</sup> Reports on Chronic Rheumatic Diseases, being the Annual Report of the British Committee on Chronic Rheumatic Diseases Appointed by the Royal College of Physicians: Number 1, edited by C. W. Buckley, M.D., F.R.C.P.; 1935. London: H. K. Lewis and Company, Limited. Royal 8vo, pp. 182, with illustrations. Price: 12s. 6d. net.

states that on one occasion, when he was sitting on the British Medical Association Arthritis Committee in 1933, the clinical classification was under discussion. A whole afternoon's voting was found to have been wrongly reported in the minutes, so the votes were taken once more; the members present at the two sessions, however, were not identical, a different set of majority votes was taken and was recorded in the report. "Counting heads" is not a scientific method of forming a decision, though it may act as a corrective to the worker who invariably wears blinkers. The book is divided into three sections. The first contains the foreword by Sir Humphry Rolleston; an account of the origin and work of the Committee on Chronic Rheumatic Diseases, By Dr. W. S. C. Copemen; and the report of the Sub-Committee on Classification and Nomenclature, by Dr. A. G. Timbrell Fisher. The second part consists of original articles. Dr. John Freeman writes on the present position of allergy and hypersensitiveness in chronic rheumatism and arthritis; Dr. Sinclair Miller makes a preliminary communication on hepatic efficiency in chronic rheumatic diseases; Dr. Joseph Race deals with biochemical investigations, Sir W. H. Willcox with focal sepsis, and Dr. C. W. Buckley with ankylosing spondylitis; Dr. A. A. Moncrieff writes on chronic arthritis in children, and Dr. W. S. C. Copeman on the place of histamine in the treatment of rheumatism. The third part of the book contains several chapters of critical commentaries and an account of the literature of the year. The titles of the chapters are as follow: "Pathological, Orthopaedic and Surgical", "The Trend of Research in Chronic Arthritis in 1934", "Nervous Manifestations in Chronic Vertebral Rheumatism", "Chronic Arthritis and its Possible Relation with the Function of the Thyroid and Parathyroid Glands".

The so-called rheumatic diseases are provisionally classified into four groups: Group I, rheumatic fever, acute or subacute; Group II, acute gout; Group III, chronic arthritis; Group IV, non-articular rheumatic affections. The third group is subdivided into the rheumatoid type and the osteoarthritic type. The rheumatoid type ("atrophic; proliferative") includes conditions of (a) specific

causation, known aetiology; (b) non-specific causation, unknown aetiology—(i) with known associated factors, (ii) with no known associated factors. It is stated that the term rheumatoid arthritis, when utilized, should be confined to conditions in the last-mentioned subdivision ("with no known associated factors"); all other forms being designated "rheumatoid type". The osteoarthritic type ("hypertrophic; degenerative") is subdivided into conditions of (a) known aetiology, (b) unknown aetiology. Detailed pathological and clinical criteria are given for the different types of Group III. One of the most interesting chapters is that on allergy and hypersensitiveness; it could well be read alone as a lesson in restraint and the need for clear thinking. Dr. Freeman shows how the general conception of allergy has altered from von Pirquet's original definition of allergy as the altered capacity for reacting which follows disease or treatment with a foreign substance: *ἄλλη ἔργα*, altered action. He suggests quite seriously that half the trouble experienced with the word allergy comes from its euphony in English—it is euphonic as compared with such words as antibody and hypersensitiveness. "Just as any young man instinctively feels that a beautiful woman must be both good and clever, so we all are inclined to believe that so beautiful a word as allergy must mean something important, without concerning ourselves too much as to what that something may be." He thinks that the word allergy, as used at present, obscures rather than helps research. In his report on liver efficiency Dr. Miller states that he found that some degree of liver impairment was present in upwards of one-third of the rheumatic cases investigated by him. After discussing biochemical investigations, Dr. Race suggests that efforts should be made to discover the reason for the great alteration in the relative amounts of albumin and globulin in the blood plasma found in chronic rheumatic diseases, though not in them exclusively. In regard to histamine, Dr. Copeman concludes that it may be a useful addition to treatment in these conditions, but he points out that much further investigation is needed to determine its sphere of usefulness. In discussing the trend of research in 1934, Dr. G. H.

Oriel expresses the opinion that what is needed is a new line of approach. "Proof that such diseases are due to a specific infection has been sought for many years, but has so far proved illusory. The study of the diathesis which may underlie the condition seems to deserve further study." He thinks that if the factors controlling the sedimentation rate of the red blood cells were discovered, a clue to the aetiology of these diseases might be found.

A few of the salient features of this most interesting book have been enumerated; it is bristling with useful information and shows clearly that many gaps still remain. In the review of the literature of the year 1934 three articles published in this journal by Australian authors are named; perhaps perusal of this book may awaken in one or more Australian investigators a desire to grapple with some of the fascinating problems with which it deals.

## Current Comment.

### EXPERIMENTAL ANÆMIA.

REFERENCE has been made in these pages to the difficulties existing in attempting to form some idea of the potency of various autanæmic preparations, that is, in assaying them for human therapeutic use. The clinical method of trial is always available, but it is obvious that it would be ideal if a reliable manufacturer could guarantee the strength of his product to be up to some predetermined standard. For this reason the experimental method is worth exploiting, but the old question arises here: are the results obtained with animals comparable with those observed in man? This criticism has been made of work carried out by Wills and Bilimoria, in which an attempt was made to establish an analogy between tropical macrocytic anæmia and the dietetic experimental anæmia of monkeys. Lucy Wills and Alice Stewart now set out to establish a parallel between the hæmopoietic arrangements of the rhesus monkey and man.<sup>1</sup> Their work is of distinct value in this field, especially in view of the previous work done by Wills on tropical anæmias and on the value of the vitamin B complex in certain of these conditions. Careful studies were made of the normal blood pictures of rhesus monkeys, which were kept under standard conditions long enough to exclude the animals suffering from malnutrition, tuberculosis and parasitic infestations. Sufficient autopsy

material was also obtained in order to establish the histological pictures of the bone marrow under normal and abnormal conditions of the blood.

Anæmia was produced in one group of animals by repeated bleedings. In man this causes a normocytic or perhaps a slightly macrocytic blood picture, soon replaced by the familiar microcytic variety of anæmia, the bone marrow showing a normoblastic hyperplasia. The response in monkeys coincided exactly with the human findings. A similar result was obtained when a chronic anæmia was produced by malarial infection. By infecting a group of monkeys with *Plasmodium knowlesi* and treating them with submaximal doses of "Atebrin", a chronic malarial state was induced, and this also gave rise to a microcytic anæmia.

Wills and Stewart then turned their attention to an anæmia of the macrocytic hyperchromic variety, which was produced in rhesus monkeys by feeding them on a diet containing very little protein and that not of animal origin, adequate in vitamins A, C and D, but deficient in B. Iron deficiency, where it occurred, was corrected. It may be noted that the diet used was based on one in common use among the poorer class of Mohammedans in Bombay, who are particularly subject to tropical macrocytic anæmia. A macrocytic and megaloblastic anæmia was produced, but the degree of variation of size in the red cells was less extreme than that usually seen in pernicious anæmia. The leucocytes were also affected, falling in number as in the human disease, while in one case a definite blood crisis was observed with large numbers of primitive red cells. *Post mortem* the body fat was almost absent; the bone marrow showed an intense hyperplastic change, but the heart and liver showed no fatty degeneration. Thus the findings differed from those of a typical pernicious anæmia. As in previous experiments with this tropical type of macrocytic anæmia, "Marmite" was found to exert a curative effect, as did also liver extract taken by mouth.

The results of this inquiry establish that the behaviour of the hæmopoietic system of the rhesus monkey strongly resembles that of the same system in man, and that this animal is therefore a suitable medium for experimental work. It is, of course, known that the tropical macrocytic anæmia is not identical with pernicious anæmia, especially with regard to the place of "Marmite" and similar extracts in treatment, but it must be remembered that other macrocytic anæmias exist in non-tropical countries, for example, the so-called acrosthic anæmia and the rare but occasional macrocytic anæmia of pregnancy. Therefore wider applications of this work are always possible.

The authors point out another interesting consideration. Miller and Rhoads, who have carried out extensive researches on experimentally produced variations in the blood picture of dogs (for example, anæmia and neutropenia), reported recently that *Bartonella canis* has been found in their animals, but only after splenectomy. The

<sup>1</sup> The British Journal of Experimental Pathology, October, 1935.

bearing of this *Bartonella canis* infection is at present obscure, and Wills and Stewart intend to observe the effect of splenectomy in monkeys in this regard. *Bartonella* is believed to be a protozoan organism, and is found in Oroya fever, which is characterized by a severe progressive anaemia. Wills and Stewart remark that in any case even if a protozoan parasite were proved to infect certain of their laboratory monkeys, it would not invalidate their work, which merely sets out to establish the value of the rhesus as a comparable animal to man in the study of anaemia. But, as they remark, the whole question of the bearing of diet upon immunity is involved, and there are many interesting possibilities in future work directed along these lines.

#### PSYCHOGENIC FACTORS IN DERMATOSES.

BLUSHING on discomfiture and sweating from fear are common phenomena, generally associated with cutaneous vaso-dilatation. In conditions of terror, however, hyperidrosis may occur with vaso-constriction. In either case there is disturbed sweat gland innervation. Angioneurotic edema may occur during or after emotional stress. Such instances illustrate the fact that mental disturbance may be accompanied by cutaneous manifestations.

F. E. Cormia and D. Slight state that the autonomic nervous system is often an intermediary channel.<sup>1</sup> They consider that autonomic imbalance may result in vagotonia—a frequent accompaniment of neuro-dermatitis. The background of rosacea includes maladjustments and nervous tensions incident to the turmoil of modern life, thus altering the sympathetic activities of the gastro-intestinal tract and leading to increased histamine liberation with peripheral vaso-dilatation. *Lichen simplex chronicus* and *prurigo nodularis* may be abnormalities of the itch mechanism due to changed sympathetic function. Neuro-circulatory instability is closely related to C. Kreibich's acute neuro-dermatitis. It is said also to be a prominent feature in many sufferers from urticaria. Nervous disorders may be lesser factors in primarily allergic diseases of the skin. Disseminate neuro-dermatitis (diathetic eczema) appears to consist of an atopic and allergic hyperirritability of the skin in over-active egocentric types. Periods of mental strain and emotional shock are often associated, by some unknown mechanism, with the onset of some dermatoses. This group includes *lichen planus*, *alopecia areata* and the "seborrhæic eczematous ear". A further group, of interest to psychiatrists and dermatologists, embraces such cutaneous lesions as paresthesias, generalized pruritus and excoriative or bizarre ulcerative changes due to wilful or unconscious actions by the patient and associated with a mental conflict.

Cormia and Slight describe a remarkable case. A man, aged twenty-nine years, displayed excoriated

papular and nodular lesions, dull red in colour, on the extensor surfaces of the upper and lower limbs and lateral aspects of the buttocks. The excoriations were so severe that superficial hæmorrhages, crusts and secondary pustulation had developed. The lesions were linear in character and sharply defined from adjacent healthy skin. The surface was smooth. In the diagnosis verrucous hyperkeratosis of *prurigo nodularis* and hypertrophic *lichen planus* were eliminated. The eruption first appeared on the anterior surface of the right leg six years previously on the scars of a healed osteomyelitis. Then the extensor surfaces of the left leg, the thighs and the upper extremities became involved. The eruption varied in severity, but was never quite absent. Multiple oozing ulcerations surrounded by narrow zones of erythema developed. There was extensive pigmentation from old lesions. *Tuberculosis cutis* was considered, but negated. The Wassermann test gave no reaction. Some of the scars were slightly keloid. The lesions were obviously results of the excoriations. There were none on the mucosæ. The patient was right-handed and the lesions were most severe on the left arm and right leg. The microscope showed acanthosis of the epidermis with hyperkeratosis rather than parakeratosis. The *stratum corneum* was denuded in many areas and in places it was separated from the *stratum granulosum* by small clusters of polymorphonuclear leucocytes. There were remissions and exacerbations, and treatment was very unsatisfactory.

Psychiatric examination disclosed the fact that the lesions began a year after marriage. There were marital difficulties and the wife was frigid. The patient did not indulge in extramarital coitus. He was of a passive and masochistic trend. Through most of his married life he had restrained his sexual urges and in such process had felt intense pruritus, which always began in the scarred area of the right leg. This region he excoriated and, as the pruritus spread, the other areas were excoriated also. This continued for thirty to forty-five minutes, when a climax came. This climax was featured by an intense burning sensation in the skin, followed by sudden relief and sleep. The relief he felt to be identical with that following coitus. The pruritus occurred only at night and when in close relationship with his wife, not when alone. No doubt marital adjustment would have had a satisfactory outcome; but the wife refused to cooperate.

Cormia and Slight consider this extraordinary case to demonstrate the intimate relation between dermatology and psychiatry. Until explained to him the patient had no idea as to the connexion between the eruption and his marital difficulties. There was no appeal for sympathy, compensation or other conscious gain which is often obvious in *dermatitis factitia*. Sexual aberrations take curious forms, and surely this instance must be unique. The climax reached by excoriation was equated by the patient with the effects of a sexual orgasm, although there was no sexual excitement or discharge. The "discharge" effect was described as a burning in

<sup>1</sup> The Canadian Medical Association Journal, November, 1935.

the skin—doubtless a vasomotor change. The result was a relief of sexual tension. The case exemplifies the transference of sexual tension to an extragenital mechanism. When the limbs were protected from excoriation the lesions rapidly involuted.

#### SUGAR TOLERANCE AND OBESITY.

THE association of obesity and glycosuria, especially in middle age and old age, has long been recognized. Certain investigators have noted that many fat people have a diminished tolerance for sugar; others have recorded cases in which obesity was associated with hypoglycæmia. This suggested that there might be a condition of hyperinsulinæmia in the early stages of obesity, followed by hypoinsulinæmia as the obesity persists. With the object of investigating this possibility, Robertson F. Ogilvie estimated the sugar tolerance of sixty-two obese persons of various ages.<sup>1</sup> Ogilvie found in this series that the longer the duration of the obesity, the lower was the tolerance for sugar. This observation was made without regard to age. It is known that sugar tolerance diminishes with increasing years; therefore Ogilvie divided his subjects into groups according to their ages, and still found that the average tolerance for sugar diminished with the duration of the obesity.

Of the subjects who had suffered from obesity for five years or less, about one-third had an increased tolerance for sugar, the remainder having a normal tolerance.

The earliest period at which diminished sugar tolerance was found was after eleven years of obesity; after eighteen years every subject in Ogilvie's series had diminished tolerance. "Tolerance ultimately becomes so deficient that the subject passes into diabetes." Ogilvie noted, in his investigation, that "diabetes supervened after periods of twelve to thirty-eight years' obesity". No relation was observed between the sugar tolerance and the degree of obesity.

Ogilvie makes some interesting observations on the influence of subnormal ovarian function on sugar tolerance. He noted that the tolerance diminished gradually with increasing years, but did not diminish at any increased rate after the menopause. On the other hand, younger women affected with ovarian hypofunction tended to have a tolerance considerably below normal. He suggests that "deficient function, premature exhaustion, or destruction of the ovaries produces a definite lowering of sugar tolerance". But he is not prepared to say whether this applies only to women who have become obese. All the subjects studied were women, excepting two. The method of estimating the blood sugar content was that of Hagedorn and Jensen.

This is a valuable contribution to the study of obesity and diabetes. But there are factors other than mere obesity that have to be considered. It is possible that obesity in some obscure way may be

responsible for diminished sugar tolerance and eventually *diabetes mellitus*; but it is more likely that the cause of the obesity is also the primary cause of these complications. Ogilvie suggests that the cause may be dietetic. Most obese persons have a badly balanced diet containing large quantities of carbohydrates; at first the islets of Langerhans hypertrophy to deal with the excess of carbohydrate foods, but later may become exhausted. Another possible factor is the disturbance of endocrine function; witness Ogilvie's remarks on diminished ovarian activity. No mention is made of the possibility of the presence of other endocrine disorders in the subjects of this investigation.

#### VITAMIN A.

PREVIOUS reference has been made in this journal to the lack of uniformity in vitamin standardization. Where chaos reigned order is gradually being brought about, largely owing to the work of the Permanent Standards Commission of the Health Organization of the League of Nations. Two conferences have been held in London under the auspices of this body, one in 1931 and the other in 1934. The decisions arrived at were the result of much research work carried out in different countries. The properties and use of the international vitamin A standard have been set out in a special report, edited by E. Margaret Hume and Harriette Chick.<sup>1</sup> This report is intended "for the information of competent workers already practised in the usual methods for the quantitative estimation of the vitamin". Our intention is merely to draw the attention of readers to the laying down of a standard; those who are interested will pursue the subject further, and technical workers will study the report. At the 1931 conference a provisional unit of vitamin A was established in terms of a sample of carotene. Soon afterwards several isomeric forms of carotene, including  $\beta$ -carotene, were isolated. At the second conference (1934) a solution of pure  $\beta$ -carotene was adopted as the permanent standard. Investigations have been carried out concerning the suitability of various oils as a solvent for  $\beta$ -carotene, and coconut oil has been found to fill all requirements. On account of the need for economizing the limited amount of  $\beta$ -carotene, it was necessary to select a subsidiary standard. After adequate tests the United States standard cod liver oil was adopted. Among the investigations recorded in the report are those dealing with a spectroscopic method of estimating vitamin A. The most important statement has to do with the necessity for the universal adoption of the international unit in place of all other units. Moreover, "satisfactory adoption of the international unit entails simultaneous comparison with the international standard in every vitamin A test".

<sup>1</sup> Reports on Biological Standards. IV: The Standardization and Estimation of Vitamin A, edited by E. Margaret Hume and Harriette Chick. Medical Research Council of the Privy Council, Special Report Series, Number 202. London: His Majesty's Stationery Office. Pp. 61. Price: 1s. net.

<sup>1</sup> The Quarterly Journal of Medicine, October, 1935.

## Abstracts from Current Medical Literature.

### MEDICINE.

#### Scarlet Fever.

J. N. HENRY (*The Journal of the American Medical Association*, August 17, 1935) reports a study of active immunization against scarlet fever by the Department of Public Health in charitable institutions and public schools of Philadelphia. A total of 10,057 children were tested, and those found susceptible by the Dick test were treated. In different groups between 26% and 58% of children tested gave positive reactions. Children between one and eight years of age gave the highest percentage of positive reactions to the Dick test, but positive reactions were quite frequent up to and over sixteen years of age. Marked reactions occurred in 4.2% of all children treated with toxin; slight reactions occurred in 30%; nausea, vomiting, fever up to 39.4° C. (103° F.), scarlet rash, sore throat and joint pains were observed. Children who did not react after the injections suffered most. Local reactions were not severe; no abscess or ulceration was noted. Among 790 children who gave positive reactions, 98% treated with scarlet fever toxin (five doses) were found to give no reaction after completion of the treatment; this failure to react persisted for one or two years at least, though seven patients among those immunized did develop scarlet fever after immunization. It is suggested that Dick testing and immunization of those found to give positive reactions should be adopted in private practice and public work, and that if this were done scarlet fever could be eliminated as a serious health problem.

#### Atherosclerosis.

T. LEAHY (*The Journal of the American Medical Association*, August 17, 1935) discusses atherosclerosis, a metabolic disease. Atherosclerosis occurs as the only form of arterial disease in the aorta and its branches, especially the coronary arteries and in the cerebral and larger renal vessels. This condition is the cause of arterial narrowing in Monckeberg's sclerosis. In youth the lesion of atherosclerosis is a fibrosis associated with lipid cells, and in older people collections of lipid cells with little fibrosis; necrosis occurs in these areas, causing atheromatous abscesses; the intima is primarily involved; thrombosis is the usual result. The lesion was found in five out of eight cases of congenital heart disease, in the left coronary artery; this favoured unusual stress as a cause. Calcification was a late stage in the process. The lipid deposit originally was formed from cholesterol, and this resulted from unsatisfactory metabolism of cholesterol. Atherosclerosis

can be produced in rabbits by feeding with cholesterol; similar results were obtained by the high fat diet given to diabetics in recent years, cream, butter and eggs being potent sources of cholesterol. Milk also is a source of cholesterol in man. Over-indulgence in milk, eggs, cream, butter and fat pork in the later half of life might account for the prevalence of atherosclerosis at that period. Ingestion of thyroid extract diminishes the amount of atherosclerosis produced in cholesterol-fed rabbits.

#### Measles.

I. M. LEVITAS (*The Journal of the American Medical Association*, August 17, 1935) discusses the treatment, modification and prevention of measles by the use of immune globulin (human). Twenty-eight patients were treated by intramuscular injection of one to two cubic centimetres of Lederle's immune globulin (human). Those patients in whom a severe racking cough or marked toxic symptoms were noted responded well to the treatment. Febrile reactions up to 40° or 40.5° C. (104° or 105° F.) were noted, but no serious symptoms occurred generally or locally. Injection of one to two cubic centimetres of globulin from one to two days after exposure to measles in eighteen cases was followed by a modified attack of measles. Further, in twelve patients of susceptible age exposed to measles, an injection of three cubic centimetres of the globulin was given for preventive purposes, with the result that none of the exposed children developed measles. Immune globulin therefore may be useful in preventing, modifying or treating measles in children. The preparation is made from human placenta, by a process which yields a fluid containing twelve milligrammes of protein per cubic centimetre.

#### Agranulocytic Angina.

WILLIAM HUGHES (*The Malayan Medical Journal*, September, 1935) reports a case of agranulocytic angina. The patient was a Eurasian woman, aged twenty-six years, who was admitted to hospital suffering from fever and tonsillitis. Scarletina antitoxin in a dose of 20 cubic centimetres was given on the day of admission. Three days later (on the seventh day of the illness), when a differential leucocyte count was made for the first time, it was found that 8% only of the leucocytes were polymorphonuclear. On the following day the total leucocytes numbered 1,100, and the polymorphonuclear leucocytes 121 per cubic millimetre of blood. By this time necrotic ulcers had appeared on the gums. On the following day there were 1,600 leucocytes, including 672 polymorphonuclear cells, per cubic millimetre. Sodium nucleinate was given (dose not stated) on this day. The leucocytes gradually increased in number to 8,800 per cubic millimetre, sixteen days after admission, and by this time the polymorphonuclear cells

numbered 6,160 per cubic millimetre. There was no history of the previous use of amidopyrine. The author remarks on the "absence of any other causal agent, except *Streptococcus hemolyticus*". He admits that it is impossible to draw any conclusion concerning the effect of scarlatina antitoxin and sodium nucleinate; but he suggests that both therapeutic measures contributed to the favourable outcome.

#### Colitis.

ALDO CASTELLANI (*The Journal of Tropical Medicine and Hygiene*, July 15, 1935) describes a type of chronic colitis that he calls "chronic metadysentery". The disorder is caused by bacilli of the "metadysentery" group, the principal pathogenic species of which are *Bacillus ceylonensis* B, *Bacillus ceylonensis* A and *Bacillus madampensis*. The author gives a description of these particular organisms as well as a short classification of the metadysentery bacilli. "Chronic metadysentery" frequently commences early in life and lasts for many years, sometimes apparently for a lifetime. It is characterized by recurrent diarrhoea with only occasional dysenteric symptoms or with none at all. The periods of quiescence may be several months or even a year or two; during them the patient may feel quite well, but usually suffers from vague abdominal discomfort, flatulence or constipation. Occasionally there may be tenderness over the sigmoid colon, the appendix or the gall-bladder. During exacerbations the stools are fluid, brownish or yellow, and at times frothy; they sometimes contain mucus, seldom blood. Fever of irregular type or low intermittent type may occur, and paratyphoid fever or malaria may be simulated. There is no tendency to spontaneous cure. For treatment the author prescribes rest, a fluid diet, and the administration of "so-called intestinal disinfectants" and a mixed vaccine of *Bacillus ceylonensis* A, *Bacillus ceylonensis* B and *Bacillus madampensis*. Bacteriophage has been tried, without any notable success.

#### Aviation.

H. G. ARMSTRONG (*The Journal of the American Medical Association*, October 5, 1935) describes the subjective mental and physical reactions to a free fall in space. In a parachute descent he jumped from an aeroplane travelling at 119 miles per hour at a height of 2,200 feet. He fell head over heels for 1,200 feet (time, eleven seconds) before opening his parachute. He noted his sensations during the fall. Having jumped out, he lost his fear and was clear-minded and observant. He was not conscious of falling until he had fallen 300 feet. With eyes closed there was a sensation of floating in space; there was no noise in the ears, as of air rushing past. There was no nausea or vertigo, breathing was easy and undisturbed. At 1,900 feet, with eyes open, a sensa-

tion of falling was noted, and this increased in intensity down to 1,000 feet, when the parachute was opened. The eyes, though unprotected, were not irritated, and vision was normal. There was a sense of rotation as the body made a complete revolution head over heels in two seconds. A feeling of pressure was noted on the downward surface of the body during the fall. The author observed that position in space and motion through space were recognized solely by means of vision; and that depth perception acuity was such that a speed of approximately 100 feet per second at a distance of 1,900 feet from the ground was required to enable him to recognize motion towards that object.

#### Poliomyelitis.

M. BRODIE AND W. H. PARK (*The Journal of the American Medical Association*, October 5, 1935) discuss active immunization against poliomyelitis. The authors state that convalescent serum is inefficacious in the treatment of preparalytic poliomyelitis. Infectious monkey cord was treated with germicides and kept for some hours to inactivate the virus. One or two doses of the prepared vaccine were given, 2.5 or 10.0 cubic centimetres being given at each dose. The vaccine was shown to be safe for monkeys, for adults and then for children. The children were tested for antibody before and after immunization; one to three doses of two to five cubic centimetres produced antibody in eight to twenty-eight days, and antibody was still present at the end of eight months. Two thousand three hundred persons received injections without significant reaction. It is suggested that vaccine might be used prophylactically for children in epidemics of poliomyelitis.

#### Anterior Pituitary Extract.

L. LOEB (*Annals of Internal Medicine*, July, 1935) discusses the thyroid-stimulating hormone of the anterior pituitary gland. He has found that a few injections of anterior pituitary extract in the guinea-pig cause changes in the thyroid gland similar to those in Graves's disease in man: loss of weight, tachycardia, protrusion of eyeballs and a rise in basal metabolism occurred in these pigs. Injection of anterior pituitary extract in female guinea-pigs produced regression of the ovarian follicles. It was further noted that continued injection of anterior pituitary in animals in which changes had been produced in the thyroid and ovaries, was followed by a return to the normal state. It was suggested that the anterior pituitary might stimulate the thyroid gland with production of Graves's disease, and that the continued action of the anterior pituitary in such a patient might lead to a remission. In the guinea-pig activity of the thyroid gland was associated with an increase of organic iodine in the blood. Inorganic iodine ordinarily stimulated the thyroid

gland, but if given with anterior pituitary it inhibited the stimulating action of the latter and thus resembled thyroid hormone, which inhibited the function of the thyroid gland. Iodine also inhibited the activity of the thyroid gland in Graves's disease, at least temporarily. Hence there was a correspondence between the effect of injections of anterior pituitary extract into guinea-pigs and the symptom complex of Graves's disease.

#### Contralateral Pneumothorax.

TOBÉ, DESGEORGES AND JOLY (*Revue de la Tuberculose*, November, 1935) assign a definite place in the therapy of pulmonary tuberculosis to contralateral artificial pneumothorax. They reserve the term for a collapse of the less affected lung induced in order indirectly to influence the other lung. It is therefore usefully employed only in the presence of a mobile mediastinum and when it appears likely that the contralateral lung will prove more retractile than the one to be made to collapse. It is, of course, far less useful than a homolateral pneumothorax, and its indications are limited and must be clearly defined. It may be performed on a perfectly sound lung, when the diseased one has been shown to be adherent, or on a less affected but still not healthy lung in similar circumstances; in the latter case there will be a double action favourable to both lungs. The lesion to be attacked should be subclavicular or juxtahilar, fairly recent, not extensively excavated, and surrounded by a fair area of comparatively sound lung. In some cases it results in complete healing of the diseased area. But even when its action is only temporary, it may be justifiable, providing a period of observation, and allowing for the making of decisions regarding further operative treatment. It may thus be possible, for instance, to substitute a partial for a complete thoracoplasty. In the performance of contralateral pneumothorax, pressures should be kept as low as possible, the lung remaining in close proximity to the chest wall. For this reason daily observation is necessary and the patient should be in a sanatorium or hospital.

#### The Effects of Repeated Large Intravenous Doses of Glucose.

H. E. HARDING (*Guy's Hospital Reports*, July, 1935) states that during some experiments on anaesthesia in rabbits it was found that animals which received intravenous injections of 50% solutions of glucose and which were given food, lost weight much more rapidly than those having no food; these animals also continued to lose weight for some days after the completion of the experiment, in spite of eating and drinking well. He has now carried out a series of experiments on unanaesthetized rabbits. The animals were given the ordinary stock diets and they received into the ear veins 20 to 25 cubic centi-

metres of a 50% solution of glucose four or five times a day until the veins were no longer available for injection. The solution tended to produce thrombosis; usually the injections were continued for three to four days. To the animals in some of the four groups into which they were divided various strengths of saline solution were given. It was found that the repeated injections were decidedly harmful to the animals if they were kept on the usual laboratory diet. The author states that the diuresis provoked by the transient hyperglycemia results in a loss of salts which cannot be replaced at all rapidly from the diet. Consequent on the loss of salts there is a dehydration of the animal's tissues, with a considerable loss of weight. Whether the tissue dehydration leads to other serious derangements of metabolism has not been determined. The author finds, however, that the loss of salt can be made good and dehydration can be prevented by substituting dilute saline solutions for tap water as a drink. The author states that the results of his experiments lend weight to the contention of Byrom that in correcting the dehydration of neglected diabetic coma saline solutions should be used instead of plain water. The author adds that when glucose solutions are administered intravenously to human subjects it would appear to be very necessary to administer salts. These salts should preferably be given by mouth in dilute solution, since by this route they are more readily utilisable.

#### Pleural Effusion During Artificial Pneumothorax.

D. MICHETTI AND A. ROULET (*La Presse Médicale*, October 16, 1935) consider that a sero-fibrinous effusion arising within six months of the commencement of artificial pneumothorax should always be evacuated, even in the absence of mechanical embarrassment or of symptoms. They exclude from this category those small collections which hardly fill the costophrenic angle and which are discovered only accidentally on screening. Effusions arising late in treatment, from eighteen months to two years, can be considered on their merits, and need aspiration only in the presence of urgent symptoms, such as dyspnoea and cyanosis. After two years the artificial pneumothorax has practically done its work, and it is not so necessary to maintain radiographic control of the subjacent lung. But in the first six months the authors insist that any appreciable effusion should be completely replaced by gas, and without any undue delay. Their two main reasons for this are: first, there is otherwise a loss of clinical and radiographic control of the underlying lung, and, secondly, pleural adhesions are likely to form, interrupting prematurely the collapse treatment before healing of the lesion can have taken place.

## British Medical Association News.

### SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Horsham District Hospital, Horsham, on November 23, 1935, Dr. R. M. DOWNES, the President, in the chair.

#### Clinical Recognition of the Arrhythmias and their Treatment.

DR. M. D. SILBERBERG read a paper entitled: "The Clinical Recognition of the Arrhythmias and their Treatment" (see page 115).

DR. G. A. PENINGTON opened the discussion by expressing his appreciation of Dr. Silberberg's most able exposition of a difficult subject. He realized that Dr. Silberberg had not attempted, in the short space of time which was available, to mention all of the arrhythmias, and Dr. Penington instanced as irregularities of no great clinical significance certain vagal irregularities which could be separated from sinus arrhythmia by the way in which they manifest themselves. In sino-auricular block an actual dropped beat occurred; there was no heart beat at the time the beat was dropped at the wrist. Other instances were bradycardia, a phasic condition without association with breathing, syncope and vagal action causing the heart's action to be at a standstill. Provided no evidence of myocardial damage existed, these dropped beats and other irregularities were not of clinical significance. The condition might bring the patient under observation and, by questioning and testing the effect of exercise, relative myocardial insufficiency might be discovered. Extrasystoles could at times be elicited by exercise followed by standing and holding the breath. Dr. Penington recalled the case of a patient with gross cardiac irregularity after an attack of sore throat, who was considered to have a myocardial lesion, possibly rheumatic. Dr. Penington had not been able to elicit evidence in favour of this view, but very frequent extrasystoles were present, sometimes amounting to halving. He had advised in this case that there was no need for treatment. Paroxysmal tachycardia sometimes presented more difficulty. He remembered a trained nurse who had consulted him because of frequent attacks of rapidity of the heart rate. She said that sometimes an attack came on suddenly, but she was sure that it did not cease suddenly. The heart was beating rapidly and regularly at about 140 beats per minute, and while Dr. Penington was auscultating he had the good fortune to hear the sudden cessation, but the patient was entirely unconscious of the onset of the condition. At times he had been able to satisfy himself that, if pressure on the right carotid sinus failed, relief from tachycardia could be obtained by pressure over the carotid sinus on the left side. The right place to apply pressure could be recognized readily, because one could feel the actual bifurcation of the carotid artery on account of the tumultuous action of the heart, and one might even feel a distinct bulging. The manoeuvre of applying pressure here was always well worthy of trial and in his experience had proved a satisfactory way of ending the paroxysm. The state of the myocardium was the key to the significance of paroxysmal tachycardia. Dr. Penington quoted the case of a man, now aged eighty-four years, who had been subject to frequent attacks of paroxysmal tachycardia from the age of fourteen years. An extra strain was thrown on the myocardium during such attacks. Paroxysmal fibrillation was the condition *par excellence* for the use of quinidine sulphate. Dr. Penington considered that this treatment was perfectly safe when the patient was a healthy young man, and in such a case he thought it was unnecessary and inadvisable to wait to see the effect of digitalis first. The thing that was most easily missed in these cases was thyrotoxicosis. When the irregular action was due to slow fibrillation, this fact could be established with more certainty by

accelerating the rate by exercise, but it was not always wise to test by exercise, and Dr. Penington thought that it was justifiable to try the administration of amyl nitrite as the means of acceleration.

Another clinical point emphasized by Dr. Penington was that oral preparations, rather than those for hypodermic injection, should be given preference in the treatment of the arrhythmias. When vomiting prevented absorption, it was often justifiable to give intravenous preparations, but great caution should be exercised in dosage because of the risk incurred of producing ventricular fibrillation. One should not hesitate to use atropine when a warning was obtained of the toxic effect of digitalis. With reference to the use of quinidine he would like to repeat what Dr. Silberberg had said about the advantage of giving the dose continuously by day and by night. As an example of failure with quinidine treatment he referred to the case of a girl with rheumatic endocarditis who had developed slow fibrillation. He attempted to restore normal rhythm by giving forty grains of quinidine daily, but was unsuccessful; the dose may have been too small; subsequently normal rhythm returned spontaneously. "Salyrgan" should be given early in the day to get the diuresis over before night-time. Dr. Penington also advocated the use of oxygen whenever cyanosis was present in these disorders, and thought that the only adequate method of administration was its continuous use, after bubbling through water, through a nasal catheter. Patients could not stand the oxygen tent. Three or four litres per hour could be given through a catheter. One should look particularly at the colour of the finger nails and the toe nails for evidence of peripheral stasis. By the use of oxygen in an efficient manner it was often possible to help the myocardium greatly to overcome a circulatory crisis.

In reply, Dr. Silberberg said that sino-auricular block and some of the other irregularities often had to be diagnosed by graphic methods. He had not been so successful as Dr. Penington in bringing about the cessation of paroxysmal tachycardia by digital pressure on the carotid sinus. He preferred the Haldane mask as the method of choice for the administration of oxygen.

DR. A. I. CHAPMAN asked Dr. Silberberg if the use of digitalis could, on occasion, lead to suppression of urine.

Dr. Silberberg said that when oliguria occurred while digitalis was being exhibited, he had been inclined to ascribe it to the effect of vomiting in reducing the intake of fluid.

DR. H. A. MAUNDER said that it was his practice when using "Salyrgan" to start with half a cubic centimetre and to give this dose and then one cubic centimetre and then one and a half cubic centimetres at intervals of twenty-four hours. With this procedure he had known the output of urine to be increased from fifteen ounces to as much as eighty ounces a day, but extreme oedema of the legs had not been relieved. He asked Dr. Silberberg if larger doses could be given with safety in such a case.

DR. WALTER SUMMONS said that he had given as much as two cubic centimetres at a time.

Dr. Silberberg said that, having established the absence of renal inefficiency, he would start with one cubic centimetre or even with two cubic centimetres. He found that the diuresis was greatly enhanced by giving a mixture containing ammonium nitrate, 20 to 30 grains, with syrup of orange and cinnamon water at four-hourly intervals for one or two days. He thought that "Salyrgan" was relatively non-toxic by comparison with "Novasurol" and had used it for as long as a year at a time. For one patient he had used it for four years.

DR. J. FORBES MACKENZIE related how he had been persuaded to see an old lady, in consultation with another doctor, because of the alarming change in her mental condition. He thought that she was "digitalis mad", and when he got the digitalis discontinued she got all right.

Dr. Silberberg commented that delirium was one of the toxic symptoms of digitalis and that it was a grave sign; he had not seen many patients recover. He would also

like to mention the toxic visual effects, such as ring vision and spinning cart wheels.

DR. LESLIE HURLEY said that in fibrillation there was a real danger in slowing the pulse rate too much. At times it was dangerous to slow it below sixty beats per minute, but it was certainly dangerous below fifty; sudden stoppage of the heart's action might result. He did not regard the onset of vomiting as a certain indication for stopping the administration of digitalis. Indeed, it might be advisable to continue it, for in some cases one could get the slowing effect only after vomiting had occurred. With reference to the massive dose method of digitalization in cases of urgency, he was satisfied that it was unnecessary to calculate the exact dose by any abstruse mathematical formula; any ordinary adult could take as much as four drachms of the fresh tincture in a single dose. It took four or five drachms at least to get digitalization. In paroxysmal tachycardia patients sometimes had little short runs of rapid heart action, and he thought that perhaps that was why they did not always notice the suddenness of the cessation of any one paroxysm. He would like to know what results Dr. Silberberg had had with regular treatment with quinidine in doses of three or four grains two or three times a day over months. Dr. Hurley had expected better results than he had obtained, for he had been only moderately successful.

In reply, Dr. Silberberg said that recurrent cases occurred and some patients might be benefited by partial thyroidectomy, even in the absence of definite signs of thyroid disease.

Dr. Hurley said that he was interested in Dr. Silberberg's remarks about the treatment of flutter, in which he had not had the same measure of success. In the treatment of extrasystoles he found that quinine, two or three grains in combination with two or three minims of *Liquor Strychnina*, had been very helpful, and in particular had given comfort to the patients in the night. It had been his practice to continue with quinidine in the treatment of fibrillation for six or seven months after normal rhythm had been restored. He recalled that Mackenzie used to get annoyed when he was referred to as the polygraph man, for Mackenzie himself always maintained that the condition of the myocardium of the ventricles was the big factor, and not the irregularity demonstrable by graphic methods, and, indeed, it was Mackenzie who had enunciated this very important truth. The outlook depended, not on the irregularity, but on the state of the heart muscle; there was a tendency to regard the irregularity as the disease.

Dr. Silberberg expressed his interest in Dr. Hurley's advocacy of quinine and strychnine for extrasystoles; he had not had much success with it himself, but would try it out again. He added some comments on the treatment of heart block. The Stokes-Adams syncope could be averted by the use of ephedrin, in a dose of half a grain three times daily. Patients subject to these attacks should carry the tablets with them for immediate use. If a doctor was called in to see such a patient during an attack, he should give a hypodermic injection of adrenaline immediately, if necessary into the heart muscle.

#### The Use of Nails in Surgery.

DR. J. G. R. FELSTEAD read a paper on the use of nails in surgery. This paper, with the discussion, will be published in a subsequent issue.

#### Thromboangitis Obliterans.

DR. GORDON FORSYTH, on behalf of Dr. Henderson, showed a male patient, aged forty-five years, a returned soldier, who had had his left leg amputated many years ago and who was subject to chronic bronchitis. The present illness had started eleven weeks before the meeting, when, after falling from a horse, the patient had felt pain in the right side and right arm; latterly pain occurred in the right leg too. No injury was revealed by radiography, the blood failed to yield the Wassermann reaction, the red cells numbered 4,300,000 and the white cells 7,500 per cubic millimetre. Since admission to

hospital the patient had had repeated attacks of pain in the fingers of the right hand and in the arm and forearm on the same side. The colour of the ring and middle fingers changed to blue, remained that colour for a few hours, then became pale, resumed the normal colour and the pain subsided. The pain during the attack was very severe and latterly was commencing to occur in the right arm as well as in the hand. At times the pulse was almost obliterated at the wrist, and during the past week or two he was getting somewhat similar attacks in the leg. Sedatives, including injections of one-third of a grain of morphine, had no effect in checking the acute pain, but he seemed to get some relief by sitting up, and considerable relief had been obtained by the use of repeated injections of "Patudin", in a dose of half a cubic centimetre night and morning.

DR. FORBES MACKENZIE said that he regarded the condition as Buerger's disease and was interested to hear of the degree of success which had been attained by injections of muscle extract.

DR. LESLIE HURLEY agreed with this diagnosis, stating that no neurological lesion could cause vascular disturbances only. The patient presented evidences of vascular obstruction; he had elicited colour changes on elevation and dependence, with persistent pallor when the hand had been contracted, and slow return on blockage of the artery on the ulnar side. Raynaud's disease was exceptionally uncommon, especially in male subjects, and he was satisfied it could be excluded in this case. He had not had much success with treatment except after sympathetic operations.

DR. M. D. SILBERBERG said that there did not appear to be much organic change in the vessels yet. Some curious vasospastic disorders had come under his notice. He had seen one man with Buerger's disease for whom amputation had been performed; later the patient had coronary thrombosis. Other patients developed transient cerebral signs. In Dr. Forsyth's patient Dr. Silberberg thought definitely that it would be advisable for him to discontinue smoking. Hot and cold applications might have at least a psychological effect.

#### Polycythæmia Vera.

DR. GORDON FORSYTH showed a married woman, aged forty-eight years, who had been admitted to hospital in March, 1934, complaining of breathlessness, pain in the left upper quadrant of the abdomen and of painful bruises which appeared spontaneously on various parts of her body. These symptoms were of many years' duration. On examination her complexion was very florid, with a cyanotic appearance of her nose and lips, and congestion of the conjunctivæ. There was an extensive bruise on the anterior portion of her right thigh and another on the dorsum of the left hand. The other positive findings were that the spleen was enlarged to a hand's breadth below the costal margin and was tender on pressure; the gall-bladder had been removed some years earlier; the heart was enlarged to 12.5 centimetres (five inches) from the mid-line, a small systolic bruit was present, and some thickening of the radial arteries could be felt; the systolic blood pressure was 210 and the diastolic 110 millimetres of mercury; the red cells numbered 11,000,000 and the leucocytes 25,000 per cubic millimetre; the urine contained a heavy cloud of albumin on boiling with dilute acetic acid, and the maximum concentration in the urea concentration test was only 2.2%. Treatment was commenced by venesection, twenty ounces of blood being removed with temporary relief; the site of venesection became extensively bruised. Phenylhydrazine hydrochloride, in a dose of one-tenth of a gramme, was given three times daily for four days and then it was discontinued. Three weeks later the erythrocyte count had dropped to 7,500,000 cells per cubic millimetre, and the systolic blood pressure to 180 and the diastolic pressure to 100 millimetres of mercury. Another similar course of phenylhydrazine was given and the patient was put on a purely vegetable diet; one month later the red cells numbered only 5,000,000, and she stated that she felt greatly improved. Since

then she had had courses of the drug at intervals of two or three months and the erythrocyte count had varied between 5,000,000 and 7,500,000, and she had shown definite clinical improvement. For the past two months phenylhydrazine had been given in the same dosage twice each week with satisfactory effect.

DR. WALTER SUMMONS issued a warning about the possible thrombosis of veins which might be the outcome of treatment with phenylhydrazine. He ascribed the death of a patient to thrombosis of the saphenous vein after the use of this drug.

DR. LESLIE HURLEY said that thrombosis was one of the recognized complications of polycythemia and that the patient to whom Dr. Summons had referred might have got the thrombosis apart from treatment with phenylhydrazine. He thought that the risk was aplastic anemia; the action of the drug might persist for three or four weeks. The cause and the pathology of polycythemia were not known; it was still regarded as an idiopathic blood disease. Repeated transfusions and Röntgenological therapy to the long bones were of value in the treatment.

DR. M. D. SILBERBERG expressed the opinion that minor grades of polycythemia were not uncommon in association with raised blood pressure. The subjects developed a characteristic dark cherry coloration, especially in the lower lids and in the retinal vessels. It had been observed that patients with typical pernicious anemia became polycythemic under intense liver therapy, and he had known patients with polycythemia who had subsequently developed pernicious anemia.

DR. G. A. PENNINGTON said that he had had a small measure of success with *Liquor Arsenicalis* as well as with phenylhydrazine.

DR. C. J. O. BROWN gave a warning against light-hearted operating on the subjects of polycythemia. He had removed a sebaceous cyst under local anaesthesia, and in the late afternoon the patient had an enormous hæmatoma at the site of the operation, which had bled for two days.

In reply to the discussion, Dr. Forsyth said that perhaps there was some relationship between pernicious anemia and polycythemia. He wondered whether an increase of the extrinsic factor led to an accumulation of extra blood cells; hence the advantages of the vegetable diet to exclude the extrinsic factor.

#### Spinal Astrocytoma.

Dr. Forsyth also showed a male patient, a railway porter, aged twenty-five years, who had complained, since July, 1935, of gradually increasing pain across the upper part of the back, in the region over the left trapezius muscle. The pain was increased by exertion and was eased only by holding his head in certain positions. The patient had noticed some numbness of the little and ring fingers of the left hand. On examination on September 12 definite weakness of the ulnar flexors of the left hand was noted and some loss of epicritic sensation over the area on the left side supplied by the seventh and eighth cervical and first thoracic segments, but there were no other localizing signs. It was seen, in skiagrams taken about this date, that the transverse processes of the seventh cervical vertebra were enlarged, and the condition was regarded as being due to a cervical rib. The occurrence of slight anal sphincteric weakness some days afterwards led to the discarding of this diagnosis in favour of a lesion of the spinal cord. By September 19 the weakness in the left forearm had increased considerably and so had the anaesthesia. At that time there was partial anaesthesia, especially to touch, heat and cold, but not to deep pressure, in the areas on each arm supplied by the lower cervical and first thoracic segments. Both knee jerks and both ankle jerks were exaggerated, the abdominal and cremasteric reflexes were abolished, and the plantar reflex on each side gave the extensor response. Ankle clonus was present. Paresis, more definite in the right inferior extremity, and partial epicritic loss, more definite on the left lower limb, were suggestive of the Brown-Séquard phenomenon. Negative results from the Wassermann test

were reported on September 18, 1935, both for the blood and for the cerebro-spinal fluid, and the cerebro-spinal fluid contained 0.208% of albumin. The fluid was not under increased pressure and the globulin content was increased. On September 19 cisternal puncture was performed and lipiodol was injected; it was shown radiographically that the lipiodol was held up at the level of the lower border of the fourth cervical vertebra. On September 27 laminectomy of the fourth to the seventh cervical vertebrae was performed under intratracheal ether anaesthesia. A diffuse type of tumour was found involving the spinal segments from the fourth cervical to the first thoracic level, affecting particularly the posterior and postero-lateral tracts and the posterior nerve roots. No line of demarcation could be found between the substance of the tumour and that of the cord. Numerous capillary hæmorrhages were scattered throughout the tumour. A small piece was excised for pathological examination. On October 10 Professor MacCallum reported from the Pathology Department of the University of Melbourne that section of the tumour showed it to be composed of tissue of nervous origin and the predominating cell was of the astrocytic type; there were numerous hæmorrhages scattered through its substance; there was no discernible demarcation of the growth from the involved tissue, though medullated fibres were seen to be involved in it; the appearances therefore suggested the diagnosis of astrocytoma, though it was impossible, of course, from the specimen to indicate the site of origin.

For about a month after the operation the patient improved, but then went steadily and progressively downhill. The left upper limb was totally paretic and almost total paresis of both lower limbs had developed with anaesthesia, almost complete up to the fourth cervical segmental level. At times the patient experienced some respiratory difficulty, and bladder symptoms had first appeared very recently.

Dr. Forsyth asked for a discussion as to the advisability of further operation in the hope that the tumour might have been extruded with more definite demarcation.

DR. LESLIE HURLEY said that apparently the tumour was an innocent type and that, as in some cases extrusion occurred, he would advise further exploration.

When asked his opinion, Dr. VICTOR HURLEY said that he would not be very hopeful.

DR. R. M. DOWNES favoured operation, hopeless as it was, because there might be further demarcation, and if even some of the tumour was removable, paralysis might be lessened with temporary benefit.

#### Traumatic Hæmatomyelia.

Dr. Forsyth also showed a young man, a shop assistant, who had been admitted to hospital on March 13, 1932. He had dived into shallow water, striking his forehead on the bottom and acutely extending his neck. He had not lost consciousness, but described a sudden loss of power in the limbs. He had been hauled out of the water half drowned. On examination the positive findings were that he had complete paralysis of all limb muscles, with the exception of some residual power in the deltoid muscles; all the trunk muscles were paralysed, the breathing being entirely diaphragmatic; the paralysis was more extreme on the left side; for several days anaesthesia was total over all areas below that of the seventh cervical segment; hyperæsthesia was elicited over the radial portions of each hand. Sensation returned rapidly on the right side and more slowly on the left. Blood-stained cerebro-spinal fluid was obtained on lumbar puncture. Sphincteric control was affected from the beginning, retention of urine necessitating catheterization for months, with incontinence of feces. Superficial reflexes were abolished, tendon reflexes were increased and the plantar response was extensor on both sides; both pupils were dilated, the left more than the right. There was no radiological or clinical evidence of bony lesion. The complications were: (i) a severe pulmonary congestion, due to the paralysis of all the accessory muscles of respiration accentuated by the partial drowning; (ii)

acute cystitis, necessitating bladder lavage; (iii) bed sores, improved by the use of an air-bed. A diagnosis was made of hæmatomyelia involving the region of the seventh and eighth cervical segments. Treatment had consisted largely of careful nursing and symptomatic treatment; splinting was used to prevent deformities; at a later stage passive and active movements were adopted; a large walking frame with straps to prevent foot-drop was found useful in getting the patient on to his feet. He had to a great extent regained the finer movements of his hands and control of the sphincters and was able to walk with a little assistance.

Dr. Forsyth thought that the points of interest were: (i) the occurrence of traumatic hæmatomyelia without bony lesion; (ii) the dilated pupils, greater on the left side, the result of interference with the cervical sympathetic; (iii) the maintenance of respiration by the diaphragm alone for several weeks; (iv) the pain over the radial aspect of the hand, enabling the irritative portion of the lesion to be localized to the seventh segment.

Dr. Forsyth added that the patient was getting vague abdominal symptoms with flatulence, which made him miserable, and asked if anyone could suggest the cause or a remedy.

Dr. G. A. PENINGTON said that it was difficult to blame the cord lesion for the abdominal manifestations, as the lesion was so high up; they might be due to some disturbance of the sympathetic control; the pains suggested a similarity to the lightning pains of tabes, for which he had been using injections of defatted milk.

Dr. LESLIE HURLEY said that the subjects of these diving injuries went on slowly improving for three or four years without any evidence of dislocation of the spine.

#### Dislocation of Cervical Vertebrae.

Dr. G. R. FELSTEAD showed a man, aged forty-four years, who had been admitted to hospital on September 24, 1935, fourteen weeks after an accident. When riding on a spring dray he had stepped on the shaft, which broke, letting him fall and causing the horse to bolt. He had been dragged one hundred yards, with the result that he was dazed and felt immediate pain and weakness in the arms; later, there was great swelling and bruising of the shoulder girdle, with severe pain in the arms and inability to raise the arms. He came to hospital holding both arms across the chest for mutual support. There was noticeable wasting of the arms, forearms and small muscles of the hands, with great loss of power. Hyperæsthesia was present over the area of distribution of the fourth and fifth and especially the sixth cervical segments; the knee jerks and ankle jerks were active and equal and the plantar reflexes gave the flexor response; sphincteric control was complete; no deformity was detectable in the cervical region, movements of the neck were almost normal and there was very slight tenderness in the mid-cervical region. It was obvious that there was a lesion of the mid-cervical segments or of the nerves in that position, and the nervous system findings alone indicated the necessity for skiagraphy. Deformity was absent to palpation, there was little or no rigidity and without special radiographic technique the anatomical diagnosis could have been missed.

Dr. Felstead demonstrated by cinema and by lantern slides of photographs and skiagrams the appearance of the arms and the range of movement of the head and neck and the fact that there was a dislocation of the fourth on the fifth cervical vertebra. On the one day two separate unsuccessful attempts had been made to reduce the dislocation by extension and a weight of one hundred pounds had been used. A plaster cast had been fitted to maintain extension. The general condition was much improved and the pain greatly diminished, and power and movement had returned to a great extent in the arms, but hyperæsthesia persisted. Dr. Felstead thought that it was of great interest that such a serious state of affairs as was revealed by radiography should have gone undiagnosed for fourteen weeks, and he asked for suggestions for further treatment and a discussion on the advisability of further attempts at reduction or of operation.

Dr. LESLIE HURLEY said that he thought that the neurological signs were due more to hæmorrhage into the grey matter than to pressure on the cord; otherwise the signs would have persisted and the pyramidal tract would have been involved. The signs extended down to the eighth cervical segment. Acute flexion of the spine would cause hæmatomyelia without the presence of dislocation. If the hæmorrhage was not very extensive, the lower motor neurone phenomena and the sensory phenomena would be limited to the upper limbs and not extend to the lower limbs. Pressure from without would disturb the long ascending and descending tracts. In Dr. Felstead's case the absence of pressure on the pyramidal tracts and the other features indicated that the condition was more one of pressure on nerve roots.

Dr. VICTOR HURLEY said that he would not attempt to do anything by open operation at the fourth cervical level. He advised continued extension with chin strap added, and the dislocated vertebra might slip into position without either the patient or the surgeon knowing when. Almost certainly a cord injury was present, extending over the lower four cervical segments, because all the signs could not be accounted for by pressure on the fourth cervical nerve roots. He would continue to support the neck in a plaster cast for a period of six months from the time of the injury.

Dr. J. FORBES MACKENZIE said that he would not advise open operation. On two occasions he had been fortunate enough to pull into position dislocated cervical vertebrae. One of the patients had been regarded as a malingerer, but on manipulation under anaesthesia he heard two loud cracks as the dislocation was reduced.

Dr. G. A. PENINGTON agreed that in Dr. Felstead's case there was a lesion of the spinal cord with involvement of the grey matter down to the first thoracic level, with weakness and wasting of the small muscles of the hands. He thought that, as two trials on the same day had been made without reduction, all that need be done now was to try continued traction, but he would not expect any amelioration of signs and symptoms, and regarded the patient's position as a precarious one.

Dr. W. R. ANGUS recalled having seen in cinema pictures that it was a custom in a certain native tribe to apply a succession of rings to the neck over a course of years until the neck became considerably elongated. He wondered whether any such procedure could be applied to Dr. Felstead's patient.

Dr. K. A. STEPHENSON considered that the state of rigidity of the muscles of the neck might have considerably reduced the effect of the pull of one hundred pounds. Böhler always used a large injection of "Novocain". With a good anaesthesia and Glisson's sling it might still be possible to make a successful reduction.

#### Oxycephaly.

Dr. Felstead also showed a patient, a girl aged thirteen years, whom he had had under observation since May, 1933, with oxycephaly. The mother had convulsions and kidney disease during the pregnancy, and noticed that the child's eyes were prominent at two and a half years. Dr. Felstead observed the presence of tower-skull. The eyes were divergent and proptosed and the approaching finger was fixed by the eyes alternately. The supraorbital ridges were flattened and the lower jaw was prognathous. There was a suggestion of webbing of some of the toes, but, contrary to the usual description of this condition, there was no choking of the disks or optic atrophy and very little visual impairment. The patient's mentality was good; she had been top of her form at school in 1932 and again in 1934. No decompression had been carried out.

#### Exophthalmic Goitre.

Dr. Felstead's next patient was a girl, aged eight years and four months, who had been brought to him for examination in June, 1933, on account of tumultuous action of the heart; prominence of the eyes had been noticed shortly before. She had obvious exophthalmos, an enlarged

thyroid, rapid tumultuous heart beat, tremor and a pulse rate of between 120 and 140 beats per minute. The systolic blood pressure was 120 and the diastolic pressure 60 millimetres of mercury. The basal metabolic rate, by Reid's formula, was +34%. On July 26, 1933, when the child was only five and a half years old, bilateral thyroidectomy had been performed. The tachycardia and tumultuous beating of the heart and the tremor had disappeared, but a slight degree of exophthalmos had persisted.

DR. VICTOR HURLEY said that the youngest subject of exophthalmic goitre upon whom he had operated was aged twelve years. He recalled the report of a congenital case in a child with a strong family history of thyroid dystrophy.

DR. H. BOYD GRAHAM said that he had seen the condition on several occasions at the Children's Hospital; the patients had not been submitted to operation, but medical measures had been adopted. He had regarded the condition as a premature appearance of the adolescent type of goitre which did not require surgical treatment but which was due to thyroid imbalance, probably associated with pubescence. Dr. Felstead had mentioned that the tumour in this case was diffuse and no doubt the symptoms had been unusually severe or Dr. Felstead would not have operated.

DR. LESLIE HURLEY mentioned the increased operative risk in this condition in a young child and that some people had advocated radiotherapy.

DR. R. M. DOWNES said that he was interested to hear from Dr. Graham that there had been cases of exophthalmic goitre in young children at the Children's Hospital, because he had never been called upon to operate on any there.

Dr. Felstead confirmed the view that the condition of this child was severe.

#### Hernia and Undescended Testicle.

Dr. Felstead also showed a boy, aged twelve years, upon whom he had carried out the first stage of the Torek operation on July 30, 1934, for left-sided congenital inguinal hernia and undescended testicle. The boy had not returned for the second stage; he was in complete comfort and without disability with the scrotum anchored to the thigh for sixteen months. At the operation Dr. Felstead found a well-developed testicle in about the middle of the inguinal canal; the funicular process was open and well defined; the sac was isolated up to the parietal peritoneum. The peritoneum, inside the abdominal cavity, was well loosened with the finger; the membranous structures about the cord were dissected and the vessels were kept intact; the gubernaculum was cut near the testicle and the scrotal cavity was opened up. An incision was made in the thigh down to the *fascia lata*; another incision was made in the lower portion of the scrotum. The posterior lip was sutured with fine chromicized gut; the testis was brought down and the gubernaculum was anchored to the *fascia lata*. The anterior lip of the scrotal wound was secured to the thigh with horsehair sutures and the hernia operation was completed.

Dr. Felstead drew attention to three special points in the operation: the clearing of the peritoneum from the abdominal wall, the dissection of the membranous structures from the cord, and the preservation of the vessels. He thought that it would be of interest to see the result of the operation and how comfortable the boy was, and he would like to get expressions of opinion as to whether the scrotum should be divided from the thigh or not.

DR. VICTOR HURLEY advised the completion of the operation by division of the scrotum from the thigh.

DR. C. J. O. BROWN congratulated Dr. Felstead on the result obtained and said that his experience of this operation was limited to about six cases, in one of which the vas had been damaged. He thought that the freeing of the adhesions was the chief difficulty and that great patience was required. In this case he advised severance of the scrotum from the thigh, not only for æsthetic

reasons, but because, as the structures were placed at present, there was an increased risk of traumatic injury to the testicle.

DR. R. M. DOWNES said that recently, at the Children's Hospital in Adelaide, he had seen a series of six patients in the various stages of completion of the Torek operation, and at a discussion there a succession of surgeons had queried the need to do the operation at all. Dr. Alan McCutcheon's statistical work in Melbourne had received wide recognition; he had observed that in Scotch College boys whom he had watched over the years, 90% to 95% of incompletely descended testicles descended, without operation, by the time of puberty. The question might be asked whether anyone had ever seen a man with two undescended testicles. It was sometimes advanced as an argument in favour of operation that there was a special liability to sarcoma in the undescended testicle, but was this not a bogey. He would make the provocative suggestion that the operation was not required, except when the undescended testicle was associated with hernia or that it was in the inguinal canal but still undescended after the age of puberty.

DR. C. J. O. BROWN spoke again and said that, in spite of Dr. McCutcheon's figures, the condition was found in adults. He had seen three examples in subjects over the age of adolescence and he had seen one instance of sarcoma of an undescended testicle.

DR. R. H. FETHERSTON said that in the vast majority of instances the presence of this anomaly was quite unknown to the boy concerned and, at times, when he had had occasion to send a note to the parents to have a boy examined by his own medical attendant because of the presence of undescended testicles, the parents had been quite frightened. He had read a translation of a German book on the potency of undescended testicle in which the author spoke of the very low fertility associated with undescended testicle, and inferred that the contents would not stand prolonged body heat; hence the anatomical arrangement in the normal person. (The ram's scrotum hung low and was kept cool and the ram was most fertile.)

#### Natural Amputation of the Appendix.

DR. W. R. ANGUS showed a specimen of an appendix in which it could be seen that the main portion was attached to the base only by a fine fibrous thread. He recounted the history and related how the appendix had been amputated naturally. The case will be reported in full in a subsequent issue.

#### Tumour of the Abdomen and Lumbar Region.

DR. STEVENS, at Dr. Felstead's request, showed a boy, aged ten years, who had come under observation in Horsham in October, 1934, on account of a lump in the back which had been noticed first in 1932. Dr. Stewart Ferguson, of Melbourne, had seen him then and several times since. The lump had been increasing in size, but no symptoms or disability had occurred. In October, 1934, there was a flattened bulge in the left loin, firm or semi-fluctuant, fixed and continuous with a tumour in the left iliac fossa. No other abnormalities were detected on systematic and repeated examinations. The systolic blood pressure was 116 and the diastolic pressure 78 millimetres of mercury. The Casoni and hydatid complement fixation tests gave no reaction. In the skiagram taken at that time could be seen the shadowy outline of a mass on the left side extending into the pelvis and up to the level of the umbilicus. At the time of the meeting, apart from a cold, the boy was well and active, and was making satisfactory progress at school and took his fair share in sporting activities. The tumour in the back overlapped the left sacro-iliac joint at the lower end and extended upwards 8.75 centimetres (three and a half inches). Medially it was closely applied to the spinous processes. It was fluctuant, but there was no hydatid thrill, no vascular hum and no impulse on coughing, and it did not transilluminate. This tumour appeared to be continuous with a tumour in the left iliac fossa, though fluctuation between the two tumours could not be demonstrated. The lower tumour extended from the level

of the umbilicus to the middle of the sacrum; though ill-defined medially, it did not appear to cross the mid-line; laterally it occupied the medial part of the false pelvis. It was firm, somewhat lobulated, fixed and not tender. On rectal examination, the lower edge was palpable as a firm elastic rounded tumour, occupying the posterior and lateral position. The spleen and liver and lymph glands were not enlarged, and there was no limitation of movement at the hip joint. The urine was microscopically clear and did not contain albumin or sugar. On examination of the blood the red cells numbered 4,600,000 and the white cells 8,100 per cubic millimetre, with a hæmoglobin value of 90%. The film was of normal appearance, and a differential white cell count showed the large lymphocytes to number 30%, small lymphocytes 5%, young polymorphonuclear cells 5%, two-lobed cells 5%, three-lobed cells 15%, four-lobed cells 20%, five-lobed cells 5%, and eosinophile cells 15%. The reports on radiography of the chest and abdomen were that the hilum shadows and vascular markings were normal, and that there was no evidence of any secondary masses in the chest; the shadowy outline of a mass extended from the transverse process of the third lumbar vertebra down to the fourth sacral level, apparently limited medially by and conforming to the edges of the vertebra; laterally it overlapped the left sacro-iliac joint. There was a depression of the left halves of the bodies of the third, fourth and fifth lumbar vertebra, which was most definite in the case of the fourth and small under-developed transverse processes of the fourth and fifth lumbar vertebra. In addition, there was a left-sided wedging of the body of the fifth lumbar vertebra with slight blurring of the edges. The surface of the ilium appeared to be intact, but the inner edge of the dorsum was thinned; a well-defined lateral displacement of the psoas muscle could be made out, and the muscle seemed to be thinned and irregularly compressed; the outline of the right kidney was normal, but that of the left kidney could not be defined.

Dr. Stevens said that he was hoping for some suggestions as to diagnosis and treatment, and Dr. Felstead said that it had been his intention to present this patient's case for discussion a year earlier, and yet very little change had occurred in the clinical course in the meantime.

Dr. H. BOYD GRAHAM said that he had noticed in a comparison of the skiagrams that in the more recent one the tumour mass appeared to be a little smaller, and the changes in the bodies of the lumbar vertebra had become demonstrable. Although he was diffident to make a diagnosis without further consideration in such an unusual case, he wondered whether it was an unusually slow psoas abscess arising from a tuberculous condition of the mid-lumbar vertebra which was undergoing absorption and had not made unfavourable progress because of the climate of Horsham and the care that had been bestowed on the boy. He suggested that dorsally the tumour was fluctuant, and that it could be needled in the expectation of obtaining fluid, the examination of which would be likely to shed light on the diagnosis.

Dr. C. J. O. BROWN said that in his opinion it was not a cold abscess; there was no calcification and the psoas muscle was shifted laterally and not hollowed out; the sheath of the psoas was distended. The negative findings for the Casoni and hydatid complement fixation tests did not by any means exclude hydatid disease, and he thought that the condition was an example of hydatid disease, and he also suggested that the tumour should be needled.

Dr. S. C. FITZPATRICK thought that hydatid disease was well worthy of consideration. Intramuscular hydatid cysts tended to elongate in the direction of the muscle fibres, and the negative result to the Casoni test did not exclude the disease. He suggested that kidney function tests and investigation of the position of the ureter by intravenous pyelography should be carried out to exclude a congenital anomaly of the kidney.

Dr. PAUL JONES said that he would like to suggest a chondroma or a myeloma of the spine as the diagnosis. He did not think that the lesion could be a cold abscess or it would have shown more signs in the last few years.

Dr. W. R. ANGUS said that he had once felt a hydatid cyst in the *gluteus maximus* muscle and thought that this tumour felt like it, with dense fibrosis around the cyst wall.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Sydney Hospital on July 18, 1935. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

#### Thromboangitis Obliterans.

Dr. GEORGE WILCOCKS showed a man, aged fifty years, who was suffering from *thromboangitis obliterans*. The patient had had pyloric stenosis associated with high acid curve in the test meal. This condition was treated by a graduated milk and milk food diet and alkaline powders every two or three hours.

He also complained of pain in the calves of both legs on walking. These symptoms were first noticed in the left leg, and as a result of gangrene occurring amputation of several toes had been performed two and half years previously. Dr. Willcocks said that the interesting point was that the condition of the legs was not the patient's main cause of complaint, and yet obviously it would soon be of the greatest importance.

On examination of the legs pulsation could not be felt in the *dorsalis pedis* arteries. The diagnosis lay between *endarteritis obliterans* and *thromboangitis obliterans*. Dr. Willcocks pointed out that opinion was divided as to the nature of these two conditions. Treatment should include the intravenous injection of fifteen ounces of hypertonic saline solution (5%) twice a week for one year and once a week for two years.

#### An Unusual Bone Disorder.

Dr. Willcocks also showed a man, aged sixty-three years, who complained of pain and stiffness in the knees. On examination there was enlargement of both knee joints and extreme mobility of the right knee joint; the upper end of the tibia could be freely moved on the lower end of the femur in an antero-posterior direction. The knee and ankle jerks were not obtained. The pupils reacted to light. It was thought that the right knee was a Charcot's joint, due to *tuberculous dorsalis*. The Wassermann test gave no reaction. The X ray report indicated Paget's disease (*osteitis deformans*) of bone. Skiagrams of various bones, including the skull, confirmed this diagnosis. However, in view of other signs, a specific affection of the right knee joint could not be excluded, and it was thought wise to advise thorough treatment with iodides.

Dr. Willcocks explained that the patient was admitted to hospital with heart failure associated with auricular fibrillation. The heart was much enlarged, and the electrocardiograph showed fibrillation of the auricles and preponderance of the left ventricle. Investigations had also revealed a blood calcium figure of 8.7. The urine calcium was 0.0935 milligramme per 100 cubic centimetres. A test meal revealed hypochlorhydria. Urea concentration in the urine was low. Treatment had been mainly directed to the condition of auricular fibrillation with heart failure.

#### Myasthenia Gravis.

Dr. WILFRED EVANS showed a male patient who was admitted to hospital on September 9, 1934, suffering from almost complete paralysis of the lower limbs, ptosis and almost complete external ophthalmoplegia. Under treatment with glycine (15 grammes) twice a day and ephedrine 0.03 gramme (half a grain) three times a day, with the addition of extra gelatine to his diet, he had made a striking recovery. His main disability was due to some contracture of the *tendo Achillis*.

#### Third Nerve Paralysis due to Syphilis.

Dr. Evans showed a man who was suffering from third nerve paralysis and absent knee jerks first noted by him in 1925. The patient had had eleven courses of "606". He still gave "+++" response to the Wassermann test, but his condition had not retrogressed.

### Pernicious Anæmia.

Dr. E. H. Stokes's first patient, a woman, aged seventy-one years, was suffering from pernicious anæmia. She had been attending the Sydney Hospital since September, 1931, when her chief complaint was that of abdominal pain, worse after food. A barium meal examination failed to reveal any organic defects.

On September 21, 1931, the result of the blood count done was as follows:

Red cells, per cubic millimetre	2,270,000
Hæmoglobin value	55%
Colour index	1.28
Leucocytes, per cubic millimetre	6,080
Neutrophile cells	53%
Eosinophile cells	1.5%
Lymphocytes	45.5%

The red cells were very irregular in size and shape. Macrocytes were very numerous. No nucleated forms were seen.

The patient was treated by means of liver extract and later by hog's stomach extract, and showed improvement, the total number of red cells rising to 4,000,000 per cubic millimetre on May 12, 1932. The treatment was continued and on January 5, 1933, she complained of a sore tongue. A blood count showed that the total number of red cells had fallen to 2,030,000 per cubic millimetre. Macrocytes were numerous. "Ventriculin" was administered, ten grammes being taken three times daily by mouth. On March 2, 1933, the total number of red cells had risen to 4,040,000 per cubic millimetre. The treatment was continued, and she appeared to do well until October 12, 1933, when a blood count showed the following result:

Red cells, per cubic millimetre	1,890,000
Hæmoglobin value	56%
Colour index	1.4
Leucocytes, per cubic millimetre	3,600
Neutrophile cells	46%
Eosinophile cells	2%
Lymphocytes	47%
Monocytes	5%

The red cells showed marked anisocytosis, very slight variation in shape; diffuse polychromasia and punctate basophilia were present; numerous macrocytes were present. No nucleated red cells were seen.

Two cubic centimetres of "Campolon" were then injected twice a week until January 8, 1934. The red blood cells then numbered 5,100,000 per cubic millimetre. A liver extract was then administered until June 7, 1934. By this time the red cells had fallen to 2,700,000 per cubic millimetre. The red cells showed considerable variation in size and shape, but the majority were large and full of hæmoglobin. Macrocytes were numerous and microcytes were also present. The administration of liver extract was continued, and on September 14, 1934, the red cells had risen to 3,230,000 per cubic millimetre. On December 8, 1934, the red cells had risen to 3,700,000 per cubic millimetre. Ten grammes of hog's stomach extract were then prescribed to be taken three times a day after meals. On March 7, 1934, 60 grammes of iron and ammonium citrate were prescribed to be taken three times a day after meals. As the patient's condition was not improving, "Campolon" was injected again, four cubic centimetres being given twice a week. The patient's condition immediately commenced to improve, and she had maintained this improvement since.

A blood count done on June 20, 1935, gave the following result:

Red cells, per cubic millimetre	5,540,000
Hæmoglobin value (12.1 grammes per centum)	88%
Colour index	0.8
Leucocytes, per cubic millimetre	6,520

The red cells showed only slight variation in size, a few cells still larger than normal, and an occasional cell smaller than normal. No macrocytes or microcytes were

seen. The cells were fairly full of hæmoglobin; no polychromasia or nucleated forms were seen. The white cells were mature. It was now proposed to continue the intramuscular administration of "Campolon", giving two cubic centimetres each fortnight.

Dr. Stokes remarked that this case illustrated the value of the parenteral administration of liver extract. There appeared to be two reasons why the oral administration was not successful. In the first place one could not be sure that the patient took the prescribed dose, and in the second place there was always a doubt as to whether absorption from the intestinal tract took place. In the "Medical Annual" for 1935 Professor Davidson had stressed the value of the intramuscular administration of liver extract. Furthermore, the cost of intramuscular therapy was considerably less than that of the oral administration of liver or hog's stomach extract.

### Pulmonary Tuberculosis.

Dr. Stokes then showed a series of patients suffering from pulmonary tuberculosis who had been treated by various methods.

One patient was a woman, aged thirty years, who had been first seen in August, 1934. She stated that she had suffered from a cough during the past two months, and had recently expectorated about a quarter of a cupful of blood. She had not lost weight or suffered from night sweats, but seven years previously had suffered from left-sided pleurisy. Physical examination showed that the movement on the right side of the chest was diminished. The percussion note at the right apex posteriorly was dull. Numerous crepitations were heard over the dull area, and the vocal resonance was increased over that region. X ray examination showed a well marked tuberculous lesion with cavitation and fibrosis. Tubercle bacilli were present in the sputum, and the blood sedimentation rate was 20 millimetres in one hour.

In September, 1934, the patient was admitted to hospital and an unsuccessful attempt was made to bring about collapse of the lung. After the patient's discharge from hospital it was decided to give her a course of injections of "Solgonal B. Oleosum". The first dose was given early in March, and she received injections at weekly intervals up till May 9. The total amount injected was 2.5 grammes of "Solgonal B. Oleosum". The patient had shown considerable clinical improvement, the cough and crepitations having almost disappeared. A report received on July 11, 1935, was as follows: "Tuberculous infiltration of the right upper and middle lobes; the condition shows little evidence of activity." It was proposed to give further injections of "Solgonal B. Oleosum".

Dr. Stokes pointed out that while it was possible that the patient might have improved with the adoption of ordinary hygienic measures, yet it seemed that the injections had had a definite beneficial effect.

The remaining patients suffering from pulmonary tuberculosis shown by Dr. Stokes had been demonstrated at the last clinical meeting of the Branch held at Sydney Hospital a year previously. They were presented again at this meeting in order to compare the various methods of treatment.

A woman, aged thirty-two years, had been treated by means of tuberculin injections. These injections had been commenced by Dr. Stokes and were now being continued by the clinical assistant, Dr. W. Bruce Fry.

Tubercle bacilli were found in the sputum on January 7, 1935, but on April 27, 1935, a report was received that after six examinations of the sputum no tubercle bacilli were found. The patient's clinical condition had shown improvement, and the blood sedimentation rate, which was 42 millimetres in one hour on August 9, 1934, was reported upon as being two millimetres on July 3, 1935. The preparation used in the treatment in this case was "T.A.F." (Bayer). The last dose given was 0.0625 cubic centimetre.

Dr. Stokes remarked that this patient appeared to be benefiting by tuberculin therapy, as the latest X ray examination had revealed considerable increase in the amount of pulmonary fibrosis.

A woman, aged twenty-six years, had been under treatment by means of pneumothorax during the past two years. Her condition had shown considerable improvement, but the latest X ray examination revealed a tendency for the lung to "creep up" from the base. Dr. Stokes pointed out that this phenomenon was frequently seen with pneumothorax therapy and constituted one of the difficulties of the treatment.

A woman, aged twenty-five years, first came to the hospital two years ago suffering from tuberculous laryngitis and left-sided pulmonary tuberculosis. The left lung had been made to collapse, but some months ago it was noticed that there were definite radiological signs of involvement of the right lung. Artificial pneumothorax had now been discontinued, and at the time of the meeting she was receiving injections of "Solgonal B. Oleosum", and appeared to have shown some improvement. Dr. Stokes remarked that the involvement of the contralateral lung occurred not infrequently during the course of artificial pneumothorax therapy, and often necessitated the reexpansion of the collapsed lung.

A woman, aged twenty-eight years, had been treated for right-sided pulmonary tuberculosis from 1929 to 1932 by means of artificial pneumothorax. Since September, 1934, she had been receiving injections of tuberculin administered by Dr. Bruce Fry. The preparation used was "T.A.F.", and the last dose given was 0.65 cubic centimetre. Dr. Stokes stated that the tuberculin therapy had been instituted in order to minimize the chance of a recurrence of the pulmonary infection.

#### Bronchiectasis.

Dr. Stokes showed a woman, aged thirty-five years, who had suffered from a cough since childhood. She expectorated one or two cupfuls of foul smelling sputum daily. She had suffered from several attacks of pneumonia. On physical examination it was noted that the right side of the chest moved less than the left. Amphoric breathing and crepitations were heard just below the right clavicle. The appearance of a skiagram after lipiodol injections suggested that bronchiectasis was present. Dr. Stokes stated that this patient was shown because the question of surgical measures such as thoracoplasty would have to be considered.

#### Bromide Granuloma.

Dr. Stokes's last patient was a woman, aged forty-six years, who had been treated for neurasthenia by means of a mixture containing fifteen grains of ammonium bromide, which was taken three times a day during a period of six months. In March, 1935, a large granulomatous mass appeared on the middle third of the left leg. Dr. Ewen Murray Will, who saw the patient in consultation, suggested that it was a bromide granuloma, and advised that the mixture be suspended.

Dr. Stokes pointed out that the bromide granuloma was not so well known as the usual acneiform type of rash produced by the bromide.

Dr. Murray Will recalled a patient seen by the late Dr. McMurray. In this case amputation of the limb had been suggested, but on the advice of Dr. McMurray the bromide was suspended and the condition cleared up.

#### Compound Fracture of the Humerus with Injury to the Radial Nerve.

DR. GEORGE BELL showed a female patient, aged eighteen years, who was admitted to hospital on November 24, 1934, suffering from a compound fracture of the left humerus and injury of the radial nerve.

The patient received a shot gun wound of the left arm on September 11, 1934. She was treated in a country hospital on a Thomas's splint for eleven weeks, and thence was transferred to Sydney.

On arrival in Sydney the limb was put into a modified Hodgen's splint with eight pounds extension, and the forearm suspended at an angle of 45°. The patient had a discharging sinus, non-union and paralysis of the muscles of the forearm supplied by the radial nerve.

On December 11, 1934, adhesions around the elbow were broken down under gas and oxygen anaesthesia. On January 3, 1935, small sequestra were removed. On March 12, 1935, the splint was removed, and the forearm and hand were put in a "cock up" splint. The arm was suspended in a sling. Massage and electricity were given.

At this time X ray films showed: (i) slow but general improvement of (a) position, (b) callus and union; (ii) the presence of foreign bodies (shot); (iii) the presence of sequestra; and at the outset (iv) periostitis.

On April 11, 1935, the patient was wearing a sling. Union was firm on clinical examination. A small amount of granulation tissue was present in the sinus. The patient was discharged to attend as an out-patient.

On May 23, 1935, X ray examination showed that union was taking place with fairly firm callus. On June 18, 1935, the patient was readmitted to hospital because as she was walking arm-in-arm she slipped and fractured the humerus at the site of the gun shot wound.

X ray examination showed the position to be good. A sinus was still present. The limb was put up on a modified Hodgen's splint. On July 18, 1935, there were no definite signs of return of function in the radial nerve. Dr. Bell said that unless recovery in function took place, the nerve should be explored as soon as the wound had been healed for about three months.

#### Syringomyelia.

Dr. Bell also showed a male patient, aged forty-one years, an Esthonian, who had been admitted to Sydney Hospital on February 25, 1932, with a perforating ulcer of the foot. The note made at the time was as follows: "Stepped on a nail six months ago with right foot. He thinks that nail penetrated skin. The patient washed the affected part and applied ointment and it healed up. Three weeks ago the part commenced to break down and patient noticed a moist spot on his sock. The hole had become larger and the discharge more copious."

On examination the patient had a perforating ulcer on the right big toe six millimetres (one-quarter of an inch) in diameter. There was no impairment of tactile sensation or of appreciation of pain. Temperature sensation was impaired over the fifth lumbar and first sacral segments. The pupils reacted sluggishly. The knee and ankle jerks were active.

An X ray examination of the right foot was made on March 2, 1932. Part of the phalanges of the second to the fifth toes had disappeared and the head and part of the shaft of the fifth metatarsal bone. There was an irregular transradiancy of the phalanges of the great toe, suggesting an osteomyelitis in these bones. The appearances in the other toes were very similar to those seen in syringomyelia. They did not resemble Charcot's joints, leprosy or Raynaud's disease. It was thought that the changes might possibly be congenital, but they were very suggestive of syringomyelia.

On March 7, 1932, spondylitis of the lower thoracic region and lower lumbar region was diagnosed. No other abnormality was seen. No reaction occurred to the Wassermann test and no bacilli of leprosy were discovered.

On April 22, 1932, the distal end of the proximal phalanx of the left great toe separated. Atrophy of the proximal phalanx of the fourth toe was present. Atrophy and separation of the distal end of the fifth metatarsal took place, as well as atrophy of the distal phalanx of the third toe on the right side. The appearances suggested syringomyelia.

On May 4, 1932, osteomyelitis of the right great toe was noted. Under ether anaesthesia the sinus was laid open and loose pieces of necrotic bone were removed. Débris was curetted away and the wound was explored. No necrotic bone was felt; the wound was plugged with iodoform gauze. On May 9, 1932, the wound was healing, and on May 20, 1932, the patient was discharged from hospital.

On May 7, 1935, the patient was readmitted to hospital with a history that the previous lesion of the foot had cleared up in six months. Three weeks before his second admission he noticed that both feet again became affected. The feet did not swell, but there was some purulent discharge. Perforating ulcers were present on the plantar

aspects of both feet. Osteomyelitis of metatarsal bone and phalanges of both feet was present.

Dissociated anaesthesia was present in both lower limbs, pain, temperature and light touch perception being lost; also the same anaesthesia was present in the right arm. There was some wasting of the muscles of the hand. The terminal phalanx of the right great toe was missing, and numerous scars were present from previous ulcers.

X ray examination on May 8, 1935, revealed well marked destruction of the head of the first, second and fifth metatarsal bones, with dislocation and destruction of the phalanges associated with all the toes. Calcification was not marked in the soft tissues, and the appearance suggested changes typical of syringomyelia. In the left foot the changes were not so advanced, with the exception of the first metatarsal, where complete destruction of the proximal phalanx had taken place. Gas was present in the soft tissues of the second toe.

On May 10, 1935, no reaction was obtained to the Wassermann and Kahn tests.

On May 14, 1935, the thickened skin and subcutaneous tissues were excised around the ulcers. An incision was made into the deeper tissues, and several small sequestra were removed. Iodoform gauze and paraffin dressing were used. The patient did not complain of pain while the above procedures were carried out.

On July 6, 1935, both feet had healed.

At the time of the meeting pain was conspicuous by its absence. There were no ocular disturbances, paraesthesiae, bladder symptoms or muscular weakness. Neurological examination revealed only disturbance of thermal sensibility on both legs and feet. Gross trophic lesions were present in both feet. Pulsation in both *dorsalis pedis* and posterior tibial arteries was felt readily and was equal. Distribution of dissociated anaesthesia was present on the feet, chiefly affecting the fifth lumbar and first and second sacral nerves.

#### Periosteal Sarcoma of Femur.

DR. LYLE BUCHANAN showed a male patient, aged nineteen years, who complained of a swelling of the right thigh of eleven months' duration, commencing in the lower third of the femur and since then involving almost its entire length. The patient's blood had not reacted to the Wassermann test. Dr. Buchanan said that the interesting features of the case were the remarkable local extent of the sarcomatous growth without obvious metastases, the textbook appearance of the skiagram, and the painless onset and cause of the disease.

#### Cancer of Head of Pancreas: Cholecystogastrostomy.

Dr. Buchanan also showed a man, aged seventy-four years, who had been referred by Dr. W. F. Smith with the following history. On April 20, 1935, the patient complained of urgent diarrhoea and some umbilical colic for two days, the diarrhoea persisting up to the time of operation. On June 4, 1935, icterus developed, painless, steadily deepening, with much loss of weight.

Clinical examination revealed severe jaundice of the obstructive type, a large tense gall-bladder, a hard craggy epigastric tumour, pyorrhoea and emphysema. The Wassermann test and tests for hydatid disease all yielded no reaction. X ray examination of the gastro-enteric tract revealed no abnormality. Occult blood was present in the faeces. The total fat was 77.8% and split fat 55% of the dried faeces. A pathological gall-bladder was found on X ray examination.

Operation was performed under spinal "Percaine" anaesthesia. The hard epigastric tumour proved to be an enormously swollen *ligamentum teres* of the liver filled with inspissated bile. Bile-stained fluid was present in the abdomen. A small (about 3.75 by 2.5 by 2.5 centimetre) tumour was present in the head of the pancreas. The common bile duct was distended to about 3.75 centimetres (one and a half inches) in diameter, the gall-bladder was large and tense, and the cystic duct was distended. Aspiration of the gall-bladder emptied the common duct. Cholecystogastrostomy was performed. The patient had an

uneventful convalescence. Icterus was much diminished, but was still present to a slight degree. The colour returned to the stool.

#### Pathological Fracture: Generalized Osteitis Fibrocystica: Parathyroidectomy.

Dr. Buchanan's third patient was a girl, aged fourteen years, who had suffered from paralysis of the left arm and forearm at two years of age; the cause was obscure. She suffered from a fractured femur at eight years of age; and she slipped in a shop on March 9, 1935, and fractured the right tibia and fibula. A pigmented capillary naevus of the face had been present since the patient's birth.

X ray examination revealed a pathological fracture and very advanced universal *osteitis fibrocystica*. On clinical examination the patient was found to be frightened, slightly resistant, and slightly anemic. The Wassermann test gave no reaction. The urine was normal and contained no Bence-Jones proteose. The basal metabolic rate was +20%. The blood calcium by various tests was found to vary from 11.0 to 9.9 milligrammes per 100 cubic centimetres, and the phosphorus from 3.4% to 3.0%. The plasma phosphatase was two units. The blood cholesterol was 131 milligrammes per 100 cubic centimetres. Other systems were normal.

Operation was performed on April 3, 1935. Both lobes of the thyroid were explored. No parathyroid tumour was found. Both right and lower left parathyroids were excised. Pathological sections of the parathyroids showed ill-defined abnormalities of growth and staining, but no definite neoplasm.

Convalescence from operation had been uneventful. The patient showed marked general improvement in physical and mental vigour. The blood calcium fell steadily over the first eight weeks from 9.8 milligrammes to 7.0 milligrammes and the phosphorus rose to 4.2 milligrammes per 100 cubic centimetres.

#### Hydrocephalus.

Dr. Buchanan's next patient was a woman, aged twenty-three years, who had complained of pains in the neck and occiput since she was ten years of age. She had a "nervous breakdown" at twelve, and at twenty with crying, headaches, giddiness and "fainting turns", inability to concentrate, and tendency to fall to the right when giddy. She was subjected to an operation in 1933 for "fibroma" of the neck, and in 1934 for chronic tonsillitis, without relief. Headaches had been of daily occurrence and incessant, very bad on waking, and intolerable on exercise, such as tennis; they were not relieved by codeine, aspirin, amidopyrin or other analgesics. X ray examination of the skull and of the cervical vertebrae, the Wassermann test, a blood count, and physical, eye and neurological examinations all gave normal results. Two septic teeth were removed without result. Autogenous blood injections, holidays, "Luminal" *et cetera*, gave no relief.

An encephalogram on February 22, 1935, showed no entry of air into the ventricles, but much increased subdural spaces over both hemispheres, and the report stated that poor filling on each side suggested bilateral meningeal adhesions, with surface brain atrophy. The provisional diagnosis was communicating hydrocephalus, probably due to birth injury. At operation on May 2, 1935, a free right subtemporal decompression was performed, so far with complete relief.

#### Premature Craniosynostosis.

Dr. Buchanan's last patient was a man, aged twenty-seven years, who gave a history of headaches since he was seven years of age, at first commencing in the afternoons and relieved by sleep, but since about 1928 continuous day and night, and greatly aggravated by exercise, stooping, noise *et cetera*, rendering the patient unable to earn a living. The result of the Wassermann test, eye accommodation, blood pressure, the ears, nose and throat, the X ray appearance of the pituitary fossa and the results of clinical and neurological examination were all normal. The patient had been edentulous since he was seventeen

years of age for alleged sepsis. Slight concentric restriction of the left field of vision only was present. Two encephalograms gave normal results, without relief of headaches. A skiagram of the skull was normal, except for "early fusion of sutures, and commencing craniostenosis".

At operation on May 18, 1932, a wide lateral cranioplasty was performed, with some removal of bone and free removal of *dura mater*. The cerebrum was under considerable pressure. Convalescence had been uneventful except for temporary numbness and incoordination of the left arm. The patient could work freely without headaches. He had been free of headaches except when worried by scarcity of work or after taking alcohol. The day before the meeting he was operated on for subacute appendicitis under narcococcal anaesthesia.

(To be continued.)

#### NOMINATIONS AND ELECTIONS.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Denning, Ben, M.B., B.S., 1929 (Univ. Sydney), Curranulla Street, Cronulla.

Stening, Samuel Edward Lees, M.B., B.S., 1933 (Univ. Sydney), Royal Alexandra Hospital for Children, Camperdown.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Broadbent, John Hayley, M.B., B.S., 1933 (Univ. Sydney), Royal Hospital for Women, Paddington.

Cowdroy, Thomas Fielding, M.B., B.S., 1933 (Univ. Sydney), c/o Dr. Allum, Cooper Street, Cootamundra.

Hall, Maida Elsie Wilhelmina Buxton, M.B., B.S., 1935 (Univ. Sydney), c/o Mr. T. G. Hall, 225 Macquarie Street, Sydney.

Harrison, Leo John, M.B., B.S., 1933 (Univ. Sydney), Bridge Street, Uralla.

Lowe, Jack West, M.B., B.S., 1933 (Univ. Sydney), 2 Dennison Hall, Marcel Avenue, Randwick.

Murphy, Eileen Mary, M.B., B.S., 1929 (Univ. Melbourne), Narooma.

Turnbull, Harley Irwin, M.B., B.S., 1929, B.Sc., 1928 (Univ. Sydney), F.R.C.S. (England), 1934, Melrose, Pritchard Street, Wentworthville.

### Public Health.

#### THE JOINT GOVERNMENT AND MUNICIPAL COMMITTEE FOR THE CAMPAIGN AGAINST POLIOMYELITIS, VICTORIA.

THE following are extracts from the report of Dr. Robert Southby, Medical Officer to the Joint Government and Municipal Committee for the Campaign against Poliomyelitis. The report is for the year ended June 30, 1935.

Total Cases: 87.

No serum: 41—

Country .. .. . 28

Metropolitan .. .. . 13

Serum: 46—

Country .. .. . 35

(a) Given with some paralysis present .. .. . 11

(b) Given before any paralysis evident .. .. . 24

Metropolitan .. .. . 11

(a) Given with some paralysis present .. .. . 4

(b) Given before any paralysis evident .. .. . 7

	Recovered (that is No Paralysis Evident at Present Time).	Improving (that is Some Residual Paralysis at Present Time).	Deaths.
Country .. .. .	5	23	0
Metropolitan .. .. .	0	12	1
(a) Given with some paralysis present .. .. .	1	10	0
(b) Given before any paralysis evident .. .. .	19	5	0
Metropolitan .. .. .	11	2	2
(a) Given with some paralysis present .. .. .	4	1	0
(b) Given before any paralysis evident .. .. .	7	0	0

#### Incidence.

During the twelve months period under review the incidence of poliomyelitis in Victoria was very slight, and would have been almost negligible, except for the occurrence of a remarkable outbreak localized almost entirely to the western district (particularly the Shires of Camperdown and Warrnambool), which commenced with a few odd cases in January, reaching its maximum in March, decreasing in April, and subsiding almost completely by the second week in May.

In all, ninety notifications were received for the State for the twelve months. In three cases the diagnosis of poliomyelitis was not substantiated by the subsequent clinical progress, thus leaving a total of eighty-seven cases for the year. Of this total forty-two comprised the western district outbreak already mentioned.

The monthly incidence was as under:

1934—	
July .. .. .	10
August .. .. .	9
September .. .. .	1
October .. .. .	Nil
November .. .. .	7
December .. .. .	4
1935—	
January .. .. .	6
February .. .. .	9
March .. .. .	21
April .. .. .	14
May .. .. .	4
June .. .. .	2

With the appearance of cases in the western district, a request was received from the District Health Officer, Dr. G. E. Cole, of Geelong, that supplies of serum should be made available where necessary.

The regular supplies at Colac and Warrnambool were increased, and fresh depôts were established at Mortlake (Dr. I. D. McInnes) and at Camperdown Hospital (Dr. E. C. Varley).

#### Serum Treatment.

A summary of the results of treatment is shown below.

It is worthy of note that the Warrnambool cases appeared generally to respond more specifically to serum treatment than those in the Camperdown area.

#### Serum Stocks.

The stocks of serum on hand as at June 30, 1935, are as follows:

#### Human Immune Serum (Antipoliomyelitis).

	Cubic Centimetres.
Metropolitan Depôts .. .. .	1,380
Country Depôts .. .. .	2,210
Commonwealth Serum Laboratories .. .. .	2,520
Total .. .. .	6,110

The distribution of the antipoliomyelitis serum (human immune) as at June 30, 1935, is as follows:

	Cubic Centimetres.
Children's Hospital .. .. .	90
Prince Henry Hospital .. .. .	70
Melbourne Hospital .. .. .	200
Town Hall, Melbourne .. .. .	560
North Melbourne .. .. .	480
Bellarat .. .. .	80
Bendigo .. .. .	120
Camperdown .. .. .	450
Charlton .. .. .	150
Colac .. .. .	290
Dimboola .. .. .	80
Echuca .. .. .	120
Geelong .. .. .	80
Hamilton .. .. .	60
Maffra .. .. .	60
Mildura .. .. .	60
Moe .. .. .	120
Mooroopna .. .. .	60
Mortlake .. .. .	140
Swan Hill .. .. .	60
Wangaratta .. .. .	100
Yarram .. .. .	140
Warrnamboul .. .. .	40
Commonwealth Serum Laboratories .. .. .	2,520
Total .. .. .	6,110

During the past six months additional supplies of both types of serum have been prepared.

#### Cooperation of Practitioners Throughout the State.

I should like before concluding this annual report to record once again my deep appreciation of the assistance of medical officers of Federal and State Health Departments, and of the support and cooperation of medical superintendents of the public hospitals, both metropolitan and country, and of practitioners throughout the State for their supply of full clinical notes of patients notified and their progress during the year.

ROBERT SOUTHBY, M.D., B.S.,  
Medical Officer of the Poliomyelitis  
Committee.

July 18, 1935.

### Post-Graduate Work.

#### COURSE FOR HIGHER SURGICAL DEGREE.

THE New South Wales Post-Graduate Committee in Medicine announces that a course for the M.S. Part I Examination, University of Sydney, will begin on Monday, July 20, 1936. Lectures will be given during the afternoon only. The course will be of approximately 12 weeks' duration, and the examination will be held at the same time as the third degree examination in November. The fee for the course will be twenty guineas, and those wishing to take the course should make application to the Secretary, New South Wales Post-Graduate Committee in Medicine, University of Sydney, from whom further particulars may be obtained.

### Correspondence.

#### CLINICAL OBSERVATIONS ON BLOOD PRESSURE.

SIR: I write with some diffidence on the subject of Dr. Southby's findings as described in his paper published in the journal of October 26, 1935.

The matter seems to me of considerable importance; we are too self-satisfied and not a little stereotyped in our views on the subject of blood pressure. Almost any middle aged woman who pours forth her tale of woe will include among her deliriums "blood pressure". How much of this is true high blood pressure and how much due to errors in our method of recording the pressure, we may learn only by thinking clearly. Dr. Southby, it seems to me, has done a great service to medicine in collecting and describing the

differences in apparent blood pressure between the two arms. I imagine that, with his paper as a stepping off point, an insight can be gained into many questions, but only if we know, for a beginning, what is the significance of the phenomena of which he writes. Without further apology I will therefore reply to Dr. Southby's letter of the journal dated December 14.

With regard to physiologists and their statements, physiologists are hardly, as a class, competent to write of clinical matters. Any general practitioner possesses more clinical data than they can ever hope to accumulate. In particular reference to Dr. Southby's argument regarding the relationship between true blood pressure and that recorded by means of a sphygmomanometer: I have myself examined a man in his nineties whose radial artery was practically incompressible. It felt, under the finger, like a string of cylindrical beads placed very close together. Pulse shock could be felt, but it was impossible to estimate blood pressure by means of a sphygmomanometer. Between the vessels of a young healthy individual and such a case there must be a regular gradation. In the youngest it may be nearly as the physiologists tell us, but the result becomes increasingly false as age advances.

Hypothyroidism is a striking example of the effects of stiffening tissues. One constantly sees women about the age of the menopause of whom, without any great clinical acumen, one could prophesy that their sphygmomanometric recording would be above normal. They tend to be constipated, fat, lethargic, depressed and sleepless. They have pains of nervous origin and are prone to operations that, like our wars, settle nothing. Their symptoms point to a low metabolic rate, while manometric readings are high. This seems anomalous, but probably the anomaly may be explained by the fact that their tissues are myxœdematous and offer an unusually high resistance to the cuff pressure. We give thyreoid and reduce the apparent blood pressure, but the effect may well be due to a lessening of the myxœdema and consequent lessening of tissue stiffness.

Dr. Southby writes of taking blood pressure with the arm relaxed. Relaxation is an end devoutly to be wished, but difficult of attainment. There are those who find it impossible to relax at the word of command, and there are some who are always relaxed. Every clinician knows how difficult it is, for instance, to examine certain abdomens, and there are some abdomens one cannot examine without the aid of an anæsthetic to relax the muscles. The same applies to arms, and between the extremes is an infinite number of variations. Being curious with regard to blood pressure in athletes, I once examined a group of amateur boxers before they went into the ring, in order to contrast the readings with those to be taken when they came out. The first readings were so erratic that I gave up the idea. The erratic results were, I think, due to excessive nervousness on the part of the subjects. One would expect such signs of strain in a group of young people about to undergo an ordeal. It was probably brought about by subconscious muscular contractions.

With regard to athletes and the study, by a Dutch physician, at the ninth Olympic games. Athletes are specialized individuals, naturally and by training; athletes of the Olympic games are highly specialized individuals. The training of an athlete is directed towards the production not of a lot of muscle, but of muscle capable of the most rapid contraction and relaxation. Velocity of movement counts for more than mere thickness of muscle. This is true even in the case of weight throwers, the distance covered by their missile being proportional to its velocity when leaving the thrower's hand. "Muscle bound" is a phrase used by trainers to indicate muscles that, though big and strong, have yet lost their capacity for rapid movement. If one examines the muscles of a trained athlete one finds them abnormally flexible; it is much easier, for instance, to define the bone beneath them than beneath those of a navvy. The navy can put forth a great deal of force, but his effort is not in the same proportion to the volume of his muscle as that of an athlete, nor is it so quick. Amongst animals the rapidity of movement of a striking snake is the nearest, in my

imagination, to the movement of a boxer's arm. Years ago Lauder Brunton showed that exercise lowered blood pressure. It is not surprising, therefore, that athletes, with their very flexible muscles and tendency towards lower blood pressure levels, do on the whole display sphygmomanometric readings lower than those of groups of ordinary mortals, though not very much lower, age for age.

Considering Dr. Southby's dynamic theory, and the whole question of the veracity of our blood pressure observations, from the mechanical viewpoint: even if one were to regard manometric readings as representing blood pressure and nothing else, it is obvious from the first that they cannot correspond with working pressure at the point of observation. Our readings are obtained by stopping the flow. When one stops the flow in the brachial artery, for example, the blood pressure becomes static, with a wave motion superposed upon it. The static portion we call diastolic pressure, the wave motion is known as pulse pressure. Considering each separately:

The diastolic pressure, due to the well-known laws of hydrostatics: on stoppage of flow, this rises to the pressure obtaining where a sufficiently large branch is given off. Probably pressure in the brachial on stoppage corresponds with that in the subclavian where the vertebral originates. This is hardly different from pressure in the aorta at origins of innominate and subclavian respectively. If this statement is near the truth and the differences between recorded pressures in the two arms are due to differences in blood pressure, the right being almost always higher than the left, flow must take place from positions of lower pressure towards positions of higher. In other words, the river must run uphill.

The pulse pressure—a wave motion arising from the thrust of new blood into the aorta by ventricular contraction. It is a wave and not a tide, as any sphygmograph will show. It is propagated, like any wave, from centre towards periphery. Nearer the centre a given section of wave is higher and contains more energy than further away. This is contrary to the direction indicated by the aforesaid differences, if they are due to differences in blood pressure.

Yours, etc.,

WILLIAM P. KELLY,

F.R.C.S. (Ireland), etc.

38 Macgregor Terrace,  
Paddington,  
Brisbane,  
December 30, 1935.

#### DOUBLE ECTOPIC PREGNANCY.

SIR: Bilateral tubal pregnancy is mentioned in several textbooks (Auspach, Eden and Lockyer). I published an instance in the *Australasian Medical Gazette*, July 20, 1912. I accept Dr. O'Connor's courteous invitation to comment, mainly to deplore that Australian surgeons, even now, are not more alive to the disastrous effect on the female economy of removing the ovaries.

Last week two young sisters came to see me. Both had had pus tubes removed two years ago; the elder one by myself. I had with some difficulty preserved both ovaries by dissecting off the pus sacs; this girl expressed herself as being very well in every way. The younger sister, as she tearfully explained to me, had suffered horribly ever since she had had her tubes and ovaries removed in a country base hospital. In her own words, "she felt her life had been ruined and that the future held out no hope for her".

Speaking generally, every gravid tube can be dissected off the ovary and nearly all pus tubes.

Yours, etc.,

RALPH WORRELL,

F.R.A.C.S., F.A.C.S. (Hon.).

233 Macquarie Street,  
Sydney,  
January 8, 1936.

#### THE X RAY TREATMENT OF ACNE.

SIR: Among many other misconceptions upon which it is quite unnecessary to comment, Dr. Raffan is clearly mistaken when he leaves it to be inferred that the brunt of my objection is to eight quarter doses of X rays in the treatment of acne. In reality, it was the advocacy of sixteen successive weekly quarter doses of X rays that compelled me to issue a warning.

It is true that I also object to the use of X rays as a routine treatment for acne, because it is often quite unnecessary, because it introduces risks which need not be taken in the great majority of cases, and because it puts the patient to needless expense. Surely these reasons are sufficient to justify objection.

As far as I am concerned, this discussion is now concluded.

Yours, etc.,

E. H. MOLESWORTH.

"Beanbah",  
235 Macquarie Street,  
Sydney,  
January 17, 1936.

### Proceedings of the Australian Medical Boards.

#### TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Kerr, George Lawson, M.B., M.S., 1893 (Glasgow),  
Repatriation Hospital, Davey Street, Hobart.  
McGregor, Alexander Hugh, M.B., B.S., 1933 (Univ. Melbourne), Launceston.  
Whitehead, James Cockburn, M.B., B.S., 1933 (Univ. Melbourne), Strahan.

#### QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1925 to 1933, of Queensland, as duly qualified medical practitioners:

Eakin, Matthew John, M.B., Ch.M., 1925 (Univ. Sydney), Brisbane.  
Henry, Allan Vernon, M.B., B.S., 1933 (Univ. Sydney), Ipswich.  
Wheelihan, John Maurice, M.B., Ch.M., 1926 (Univ. Sydney), Allora.

### Obituary.

#### DENIS FRANCIS BARRETT.

WE regret to announce the death of Dr. Denis Francis Barrett, which occurred on December 21, 1935, at Clermont, Queensland.

#### GERALD EUGENE MACDONALD STUART.

WE regret to announce the death of Dr. Gerald Eugene Macdonald Stuart, which occurred on January 7, 1936, at Rockhampton, Queensland.

## Books Received.

- AN APPLE A DAY, by Sir W. Arbuthnot Lane, Bt., C.B.; 1935. London: Methuen and Company, Limited. Crown 8vo, pp. 184. Price: 5s. net.
- A TEXTBOOK OF BACTERIOLOGY, by T. B. Rice, A.M., M.D.; 1935. Philadelphia and London: W. B. Saunders Company; Melbourne: W. Ramsay (Surgical) Proprietary Limited. Royal 8vo, pp. 551, with illustrations.
- ANTENATAL AND POSTNATAL CARE, by F. J. Browne, M.D., D.Sc., F.R.C.S., F.C.O.G.; 1935. London: J. and A. Churchill. Demy 8vo, pp. 495, with 58 illustrations. Price: 15s. net.
- CATECHISM SERIES: PHYSIOLOGY; Fourth Edition; Parts III, IV and V; 1935. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 389.
- CATECHISM SERIES: HISTOLOGY; Third Edition; 1935. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 76.

## Diary for the Month.

- FEB. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
- FEB. 5.—Western Australian Branch, B.M.A.: Council.
- FEB. 5.—Victorian Branch, B.M.A.: Branch.
- FEB. 6.—South Australian Branch, B.M.A.: Council.
- FEB. 7.—Queensland Branch, B.M.A.: Branch.
- FEB. 11.—New South Wales Branch, B.M.A.: Executive and Science Committee.
- FEB. 14.—Queensland Branch, B.M.A.: Council.
- FEB. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
- FEB. 19.—Western Australian Branch, B.M.A.: Branch.
- FEB. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
- FEB. 26.—Victorian Branch, B.M.A.: Council.
- FEB. 27.—South Australian Branch, B.M.A.: Branch.
- FEB. 28.—Queensland Branch, B.M.A.: Council.

## Medical Appointments.

Dr. F. J. Short has been appointed Government Medical Officer at Nambour, Queensland.

Dr. A. R. S. Vickers has been appointed Quarantine Officer at Port Hedland, Western Australia.

Dr. W. C. T. Upton has been appointed Honorary Dermatologist, with the status of Honorary Assistant Physician, at the Adelaide Hospital, South Australia.

Dr. L. W. Linn has been appointed Honorary Clinical Assistant at the Adelaide Hospital, South Australia.

## Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," pages xvi, xvii, and xix.

- BROOKTON HOSPITAL COMMITTEE, BROOKTON, WESTERN AUSTRALIA: Medical Officer.
- COMMONWEALTH DEPARTMENT OF HEALTH: Medical Officer.
- MENTAL HOSPITALS DEPARTMENT, PERTH, WESTERN AUSTRALIA: Assistant Medical Officer.
- RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Honorary Assistant Dermatologist.
- ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.
- ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Senior Resident Medical Officer.
- THE UNIVERSITY OF MELBOURNE, VICTORIA: Beane Scholar-ship in Pathology, Part-time Lectureship in Pathology.
- THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, SYDNEY, NEW SOUTH WALES: Resident Medical Superintendent, Medical Registrars.
- VICTORIAN EYE AND EAR HOSPITAL: Resident Surgeons.
- WESTERN AUSTRALIAN PUBLIC SERVICE: Medical Officer of Schools.
- WONGAN HILLS DISTRICT HOSPITAL BOARD, WESTERN AUSTRALIA: Medical Officer.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Richmond District Hospital, North Queensland. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY Hospital, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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